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Vol. XCII, N. S. Vol. 45 MAY, 1938 No. 5

ARE MORE COLLEGES NEEDED?

Interest in the question whether this country has enough veterinarians has shifted to a consideration of whether we have enough veterinary colleges. It is no secret that about half of the colleges in the country have been turning students away for several years. Nor is it any secret that many of these students who have been declined admission, first at one college, then at another, have been welcomed with open arms at some of the other institutions. Whether any permanent danger attends this situation remains to be seen. A few years ago, some of our colleges were suffering from inanition, due to the small number of students in attendance. Now some of these same colleges are showing symptoms of indigestion, due to engorgement. What was a famine is now a feast.

With an equitable distribution of students among the ten veterinary colleges in the United States, and an even division of these students among the four classes, it is believed that the ten colleges could graduate 400 veterinarians each year without any difficulty. This is 100 more than the estimated requirement reported by the A. V. M. A. Committee on Education about eight

years ago. It will be recalled that this estimate made no provision for any enlargement of the field of veterinary medicine that would require more veterinarians. Since the report referred to was made, the only important extension of veterinary service has been in the Army, in connection with food inspection in the CCC camps. This work has called a hundred or so veterinarians into active service in the Veterinary Reserve Corps.

It is true that federal-state projects, aimed at the eradication of bovine tuberculosis and the control of Bang's disease, have absorbed a large number of veterinarians during the past few years. But this has been a temporary condition. There is absolutely no guarantee that it is permanent. It is a condition that could change on very short order if federal funds were to stop flowing from Washington. In some states, where practitioners were not permitted to conduct this work in their own territories, private practice has been hurt very materially and it will take the profession years to regain the lost ground.

We hear much concerning the activities of veterinarians in the field of public health. A comparatively small percentage of veterinarians have qualified to participate in public health work. Just because a man has a degree in veterinary medicine does not qualify him for public health work any more than a degree in medicine qualifies a physician. Graduate work is necessary in both cases. Then, too, the salaries which now go with these positions, except those in the administrative brackets, are not particularly attractive to veterinarians. In this respect the present situation has been likened to the dog chasing its tail. On the one hand, we hear arguments that more veterinarians are needed to fill positions, and on the other hand that more positions are needed to absorb an increasing number of veterinarians.

Perhaps, after all is said and done, the only reason why we hear any talk of a need for more veterinary colleges is because a number of prospective veterinary students have been declined admission by the college of their choice. It is true that several of our large agricultural states are without veterinary colleges. It is equally true that some of the colleges take care of the applicants from their respective states first. This makes it hard for the young men in states without colleges, who have their eyes on veterinary medicine as a career. But would it be good business for these states to start veterinary colleges to take care of these cases? We believe not. We rather lean to the belief that what we need is better trained veterinarians rather than more poorly supported veterinary colleges. It takes a lot of

money to equip and run a veterinary college these days, and few states have shown any inclination to do the job the way it ought to be done.

Diamond Jubilee Convention
Hotel Pennsylvania—New York
July 5-6-7-8-9, 1938

EXECUTIVE BOARD ELECTIONS

The regular elections from members of the Executive Board in District 2 (Pennsylvania, New Jersey and Delaware) and in District 3 (Indiana, Illinois and Wisconsin) are now in progress and will come to a close during the present month. The polls for the election in District 3 will be closed on May 10, but owing to the unavoidable delay in sending out the ballots for the election in District 2, the polls for that election will not be closed until May 18. The members of the Executive Board who are successful in these elections will take office at the close of the meeting in New York, to serve for regular terms of five years each.

Diamond Jubilee Convention
Hotel Pennsylvania—New York
July 5-6-7-8-9, 1938

PLANS

The June issue of the JOURNAL will be a special number, published in the interests of the Diamond Jubilee Convention of the American Veterinary Medical Association, which will be held at the Hotel Pennsylvania, in New York City, July 5-9. It will be the most elaborate convention number of the JOURNAL ever attempted and, in addition to going to our regular mailing list of more than 5,200, a copy will be mailed to every graduate veterinarian in the country who is not a member of the A. V. M. A., and for whom we have an address. This special number should not be confused with the revamped JOURNAL which will make its appearance in July.

FINANCIAL REPORT

JANUARY 1, 1937, to DECEMBER 31, 1937

Bank balance, December 31, 1936			$ 3,844.84
Receipts for Calendar Year			
By transfer from Salmon Memorial Fund to A. M. A. Fund		$ 400.00	
Received from Secretary			
A. V. M. A. Fund	$14,284.41		
JOURNAL Fund	26,511.85		
Exhibit Fund	1,180.00		
		41,976.26	
Interest on U. S. Government Bonds			
A. V. M. A. Fund	$ 1,076.11		
JOURNAL Fund	136.11		
A. V. M. A. Special Fund No. 2	137.50		
		1,349.72	43,725.98
Total receipts			$47,570.82
Disbursements for Calendar Year			
A. V. M. A. Fund	$20,911.46		
JOURNAL Fund	22,558.43		
A. V. M. A. Special Fund No. 2	137.50		
Total disbursements			$43,607.39
Bank balance, December 31, 1937			$ 3,963.43
Revolving Fund in hands of Dr. Hoskins			500.00
Total cash resources			$ 4,463.43
U. S. Government Bonds ($41,000.00 par value) at cost			41,259.02
			$45,722.45
Less: Amount due Exhibit Fund 1938			1,180.00
Total assets December 31, 1937			$ 44,542.45
Total assets December 31, 1936		$54,114.36	
Less: Bonds belonging to other Funds — Salmon Memorial Fund ($3,000.00 par) cost	$ 3,041.44		
A. V. M. A. Special Fund No. 2 ($5,000.00 par) cost	5,069.06	8,110.50	46,003.86
Decrease in assets for year			$ 1,461.41

DISTRIBUTION OF ASSETS

Fund	Cash	Bonds at Cost	Total
A. V. M. A.	($26,109.77)	$ 5,821.94	($20,287.83)
JOURNAL	29,393.20	35,437.08	64,830.28
Totals	$ 3,283.43	$41,259.02	$44,542.45

Respectfully submitted,
(Signed) M. JACOB, *Treasurer.*

APPLICATIONS FOR MEMBERSHIP

(See January, 1938, JOURNAL)

FIRST LISTING

ALEXANDER, JAMES M. Cannon Falls, Minn.
D. V. M., McKillip Veterinary College, 1913
Vouchers: Joab P. Foster and William C. Prouse.

BEAN, CLYDE W. 2304 Belmont Blvd., Nashville, Tenn.
D. V. M., Colorado State College, 1935
Vouchers: Hugh L. Fry and Elmer B. Parker

BECKER, HENRY C. 24 W. 109th Pl., Chicago, Ill.
M. D. V., McKillip Veterinary College, 1911
Vouchers: L. A. Merillat and H. Preston Hoskins.

BURCH, CLARK W. Box 2, Phillips, Wis.
B. S., Kansas State College, 1932
D. V. M., Kansas State College, 1937
Vouchers: R. R. Dykstra and James S. Healy

DESSON, LEONARD J. Linden Rd., Brighton Sta., Rochester, N. Y.
D. V. M., Cornell University, 1920
Vouchers: C. P. Zepp and J. J. Merenda.

FREMONT, MARTIN H. 2500 16th St., San Francisco, Calif.
D. V. M., Cornell University, 1936
Vouchers: Joseph M. Arburua and John McInnes.

GSELL, PRESTON L. 932 Venice Blvd., Los Angeles, Calif.
V. M. D., University of Pennsylvania, 1934
Vouchers: W. L. Curtis and L. B. Wolcott.

HEATH, C. LESTER Billings, Mont.
D. V. M., Colorado State College, 1933
Vouchers: Jas. Farquharson and A. A. Hermann.

HOFFMAN, OSCAR A. Box 113, Walnut Grove, Calif.
D. V. M., San Francisco Veterinary College, 1915
Vouchers: Joseph M. Arburua and John McInnes.

HUBBARD, HARRY E.
Department of Agriculture and Markets, State Capitol, Madison, Wis.
D. V. M., Kansas State College, 1937
Vouchers: W. Wisnicky and J. E. Weinman.

KINSEY, GERTRUDE FISHER 154 W. 11th St., New York, N. Y.
D. V. M., Cornell University, 1936
Vouchers: C. P. Zepp and E. Sunderville.

LAUGHLIN, WESLEY M. Burton, Ohio
B. V. Sc., Ontario Veterinary College, 1919
Vouchers: R. R. Laughlin and F. A. Zimmer.

LOVE, FORREST C. Department of Agriculture, Sacramento, Calif.
D. V. M., Kansas State College, 1931
Vouchers: W. L. Curtis and E. M. Austin.

MCGRATH, WILLIAM E. 999 Exchange Ave., Chicago, Ill.
V. S., Montreal Veterinary College, 1888
Vouchers: Israel Wallman and B. H. Steigleder.

MARKHAM, FREDERICK D. C. 345 W. 2nd St., Elyria, Ohio
D. V. M., Cornell University, 1935
Vouchers: N. D. Backus and H. N. Umbstaetter.

MENIG, BERT A. Department of Agriculture, Olympia, Wash.
B. S., D. V. M., State College of Washington, 1937
Vouchers: E. E. Wegner and J. E. McCoy.

POMEROY, BENJAMIN S. 880 Linwood Pl., Saint Paul, Minn.
D. V. M., Iowa State College, 1933
Vouchers: Joab P. Foster and William C. Prouse.

PYLES, RAY S. Box 422, Manhattan, Kan.
D. V. M., Kansas State College, 1937
Vouchers: Edwin J. Frick and Samuel Kelsall, III.

SPENCE, ELTON M. 769 Vance Ave., Memphis, Tenn.
D. V. M., Alabama Polytechnic Institute, 1937
Vouchers: John H. Gillmann and E. B. Mount.

THORP, WILLIAM T. S. Michigan State College, East Lansing, Mich.
D. V. M., Michigan State College, 1935
Vouchers: E. T. Hallman and B. J. Killham.

WINWARD, JOHN D. 611 S. 8th East, Salt Lake City, Utah
B. S., D. V. M., State College of Washington, 1932
Vouchers: W. H. Hendricks and Jean C. Flint.

Applications Pending

SECOND LISTING

(See April, 1938, JOURNAL)

Cain, Harold W., 17422 Woodward Ave., Detroit, Mich.
Cameron, James A., Horse Infirmary, Union Stock Yards, Chicago, Ill.
Collier, James R., 3441 Garretson Ave., Sioux City, Iowa.
Deal, Blakey T., 5350 Webster St., Philadelphia, Pa.
Dunn, William T., Box 428, Hartsville, S. C.
Hamner, Robert H., 409 Post Office Bldg., Montgomery, Ala.
Hinkle, Thomas C., Jr., Caseyville, Ill.
Kelly, James J., Marshall, Minn.
Kitchen, George R., Sumter, S. C.
Lavitan, Murray J., c/o City Health Dept., Charlotte, N. C.
McNeely, Frank F., Colfax, La.
Manthei, Chester A., 904 Acklen Ave., Nashville, Tenn.
Michael, Lloyd J., 1805 Church St., Cleveland, Tenn.
Moir, Thomas A., 1249 Morningside Ave., Sioux City, Iowa.
Molengraft, Joseph A., 6317 S. Mozart St., Chicago, Ill.
Neidig, Charles C., Aitkin, Minn.
Riley, Emmett T., 336 Post Office Bldg., Oklahoma City, Okla.
Rode, Walter F., White Bear, Minn.
Schubert, Carl J., Slinger, Wis.
Stephenson, Hadley C., 105 Cornell St., Ithaca, N. Y.
Stevens, John D., Box 852, Sequim, Wash.
Sutton, Karl L., Morenci, Mich.
Titterud, Oscar H., Preston, Minn.
Venzke, Walter G., Galva, Iowa.
Wentworth, James E., Rochester, Minn.
Young, Anderson V., Route 1, Box 167, Shreveport, La.

The amount which should accompany an application filed this month is $8.33, which covers membership fee and dues to January 1, 1939, including subscription to the JOURNAL.

Diamond Jubilee Convention
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COMING VETERINARY MEETINGS

Ak-Sar-Ben Veterinary Medical Association. Fontenelle Hotel, Omaha, Neb. May 9, 1938. Dr. J. D. Ray, Secretary, 1124 Harney St., Omaha, Neb.

Hudson Valley Veterinary Medical Society. Catskill, N. Y. May 11, 1938. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Willamette Valley Veterinary Medical Association. Tillamook, Ore. May 14, 1938. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Mississippi Valley Veterinary Medical Association. Galesburg Horse and Mule Barn, North Cherry St., Galesburg, Ill. May 26, 1938. Dr. Lyle A. Gray, Secretary, Bushnell, Ill.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. June 1, 1938. Dr. J. B. Engle, Secretary, Box 432, Summit, N. J.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R. St., Detroit, Mich. June 1, 1938. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Dallas-Fort Worth Veterinary Medical Society. Fort Worth, Texas. June 2, 1938. Dr. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. June 2, 1938. Dr. E. G. Pigman, Secretary, 4206 Polk Ave., Houston, Texas.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. June 7, 1938. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Midwest Veterinary Conference and Clinic. Municipal Auditorium, Kansas City, Mo. June 7-9, 1938. Dr. S. J. Schilling, Corresponding Secretary, Box 167, Kansas City, Mo.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. June 8, 1938. Dr. J. P. Torrey, Secretary, 610 Veronica Ave., East Saint Louis, Ill.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. June 14, 1938. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

Ohio State University Veterinary Conference. Ohio State University, Columbus, Ohio. June 15-17, 1938. Dr. Oscar V. Brumley, Secretary, Ohio State University, Columbus, Ohio.

Idaho Veterinary Medical Association. Boise, Idaho. June 17-18, 1938. Dr. E. M. Gildow, Secretary, University of Idaho, Moscow, Idaho.

Georgia State Veterinary Medical Association. Columbus, Ga. June 20-21, 1938. Dr. J. E. Severin, Secretary, 357 Edgewood Ave. S.E., Atlanta, Ga.

Oklahoma Veterinary Medical Association. Hotel Tulsa, Tulsa, Okla. June 20-21, 1938. Dr. F. Y. S. Moore, Secretary, McAlester, Okla.

Eastern Iowa Veterinary Association, Inc. Dr. F. M. Wilson's Hospital, Mechanicsville, Iowa. June 21, 1938. Dr. John J. Strandberg, Secretary, 1005 Eighth Ave., Belle Plaine, Iowa.

South Carolina Association of Veterinarians. Charleston, S. C. June 23-24, 1938. Dr. R. A. Mays, Secretary, 415 State Office Bldg., Columbia, S. C.

North Dakota Veterinary Medical Association. North Dakota Agricultural College, Fargo, N. Dak. June 27-28, 1938. Dr. M. C. Hawn, Secretary, 310 Thirteenth St. N., Fargo, N. Dak.

Wyoming Veterinary Medical Association. Laramie, Wyo. June 27-28, 1938. Dr. H. D. Port, Secretary, 304 State Capitol Bldg., Cheyenne, Wyo.

California State Veterinary Medical Association. Yosemite National Park, Calif. June 27-29, 1938. Dr. Chas. J. Parshall, Secretary, Brentwood, Calif.

American Association for the Advancement of Science. Ottawa, Ont. June 27-July 2, 1938. Dr. F. R. Moulton, Secretary, Smithsonian Institution Bldg., Washington, D. C.

Michigan State Veterinary Medical Association. Michigan State College, East Lansing, Mich. June 28-29, 1938. Dr. H. H. Clark, Secretary, 2601 E. Michigan Ave., Lansing, Mich.

North Carolina State Veterinary Medical Association. Robert E. Lee Hotel, Winston-Salem, N. C. June 28-29, 1938. Dr. Pascal C. McLain, Secretary, Route 4, Charlotte, N. C.

Virginia State Veterinary Medical Association. Hotel Patrick Henry, Roanoke, Va. June 30-July 1, 1938. Dr. A. J. Sipos, Secretary, 1102 State Office Bldg., Richmond, Va.

Wisconsin Veterinary Medical Association. Voss' Birchwood Lodge, Manitowish, Wis. June 30-July 1, 1938. Dr. B. A. Beach, Secretary, University of Wisconsin, Madison, Wis.

American Veterinary Medical Association. Hotel Pennsylvania, New York, N. Y. July 5-9, 1938. Dr. H. Preston Hoskins, Secretary, 221 N. LaSalle St., Chicago, Ill.

MENINGITIS, ENCEPHALITIS AND MYELITIS IN DOGS*

By CARL F. SCHLOTTHAUER, *Rochester, Minn.*
Division of Experimental Medicine, The Mayo Foundation

Observations by veterinarians and others throughout the world tend to indicate that meningitis, encephalitis and myelitis occur frequently in dogs. Unfortunately, these disease processes are too often incorrectly designated as "running fits," "barking fits," or as various other kinds of fits or paralysis according to the symptoms manifested. Undoubtedly most of these conditions are due to primary or secondary disease of the central nervous system, but the names given them do not indicate this. Because of the apparent high incidence of these diseases in dogs, I wish to discuss some of them, briefly paying particular attention to encephalitis. Some cases will be reported to illustrate the symptoms and lesions.

The term "encephalitis" is used to designate inflammatory disease of the brain. The disease process may be acute, subacute, or chronic, and it may be focal or disseminate, simple or purulent. If the inflammatory process has its origin in the meninges and extends along the blood-vessels and lymph-spaces into the brain, it is termed "meningo-encephalitis." Likewise, if the brain and spinal cord are affected simultaneously, the disease process is termed "encephalomyelitis." Not infrequently two or more parts of the brain are affected at the same time.

ETIOLOGY

The etiology of meningitis, encephalitis and myelitis is various. Probably viruses and bacteria and their toxins are the chief causes of inflammatory and destructive lesions, although many organic and inorganic substances will also cause irritation and destruction of nerve tissue. Because of that, the designation of the causative agent of various nervous diseases is not simple.

Various bacteria and viruses are known to cause disease of the central nervous system, and the disease produced may be primary or secondary in the nervous system. Nervous complications occur with many diseases; and they are frequent with canine distemper. Epileptiform convulsions frequently are an early symptom of distemper in young dogs, and some investiga-

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

tors are of the opinion that canine distemper may manifest itself as a primary disease of the central nervous system. However, many of those who are investigating canine distemper are of the opinion that the nervous symptoms are due to a secondary disease process.

Lockhart and Johnson,[1] for example, in their observations made on several thousand healthy young dogs exposed to distemper, mentioned that they had never observed the occurrence of so-called "distemper spasms" before the lapse of at least 21 days following definite exposure to distemper. These authors made stained preparations from the pia mater and from the cerebrospinal fluid obtained from the lateral ventricles of dogs affected with the so-called nervous forms of distemper. They found peculiar organisms, diplococci or streptococci, to be quite constantly present. However, this is not definite proof that these organisms are the causative agents of the disease.

Encephalitis has been observed to occur as a primary disease in both young and adult dogs, and the presence of a previous disease has not been noted in such cases. In similar cases in which I carried out studies on necropsy material, I failed to find definite organisms in the brain and spinal cord, although cell inclusion bodies were observed in some of the motor cells of the brain and spinal cord in one case of acute encephalomyelitis. This absence of organisms may have been attributable to the fact that the disease process was chronic in most of my cases.

Various food, fat, vitamin and mineral deficiencies have been credited with causing certain nervous diseases in animals. Some nervous diseases have been definitely proved to be associated with certain diets. We know that disturbed calcium metabolism will cause tetany-like symptoms in animals. Baashuus-Jessen,[2] in a discussion of certain nervous diseases of dogs in the Arctic, mentioned fat-deficient diets as the chief cause of these ailments. It is thought that when all dispensable fats are exhausted, a drain of the glycerin-phosphoric acid in the lecithin of the central nervous system takes place with ensuing nervous disorders. Baashuus-Jessen also mentioned that loss of fat may cause disturbances of calcium metabolism, and he stated that if sufficient fat is provided in the diet these diseases do not occur.

Green[3] observed hemorrhagic encephalitis in foxes that were receiving a certain fish diet. The hemorrhages occurred chiefly in the brain stem and medulla and caused spastic paralysis. Two similar cases were observed by me. Uremic meningitis is known to occur in certain cases of marked uremia; I observed a case

of spontaneous uremic meningitis in a wild gray squirrel. Hypoglycemia causes definite nervous symptoms. This disease is observed chiefly in man and it usually is caused by an overdose of insulin. However, spontaneous hypoglycemia was observed in wild snowshoe rabbits by Green[4] and his associates in Minnesota.

Intestinal parasites are known to cause tetany-like convulsions in puppies and in some adult dogs. Wells[5] recently reported the occurrence of paralysis in dogs infested with ticks (*Dermacentor variabilis*). Prompt recovery was noted when the ticks were removed.

In man, diseases of the nervous system frequently are secondary to blood-vascular diseases and syphilis. Fortunately, blood-vascular diseases are infrequent in lower animals and syphilis does not, of course, occur.

Symptoms

The symptoms of disease of the central nervous system may be general or localized. General symptoms may be caused by diffuse disease of the cortex of the brain, with or without an associated increase in intracranial pressure, or by an increase in the intracranial pressure alone. Localized symptoms as a rule are associated with focal lesions in the brain or spinal cord which interefere with certain functions.

It is not always easy to differentiate meningitis and encephalitis in lower animals. Some symptoms manifested are seen in both diseases and both may rapidly terminate in coma and death.

Meningitis: Meningitis usually causes hyperesthesia about the head and neck. Deep palpation of the skull and muscles of the neck causes discomfort and pain. Marked tonic spasms of the muscles of the face and neck are frequent. The head may be drawn up and backward. Affected animals appear to dislike to move the head and will violently resist attempts to move it forcibly. The temperature may be elevated and the pulse accelerated, but it is not uncommon to observe a normal pulse rate in the presence of an elevated temperature. Affected animals appear restless and irritable, and sudden loud noises may cause marked excitement and localized or general spasms.

Epileptiform convulsions are frequently observed, but they are more common in young animals than in adults. Vomiting is a frequent symptom in acute meningitis. The pupils usually are contracted early in the course of the disease, but may dilate later; pupillary reflexes may be absent or unequal. Examination of the fundus frequently reveals hemorrhagic retinitis and papil-

lary neuritis. Impairment of vision is not infrequent. The patellar and foot-pad reflexes usually are unchanged. As the disease progresses, mental disturbances, such as abnormal excitement or depression and stupor, are noted.

If the spinal meninges are affected, as in pachymeningitis for example, the animals are loath to move and when they do so they hold the body rigid and move slowly. They have difficulty in arising when lying down. Early in the course of the disease the legs may be extended and rigid, and the back is arched. This probably is to eliminate movement of the spine. Hyperesthesia over the back is marked. The condition frequently results in muscular atrophy. Bone deposits in the form of platelets are not uncommon in pachymeningitis, and may eventually impinge the spinal nerves and cause peripheral paralysis.

Encephalitis: Encephalitis may cause general or localized symptoms, depending on whether the disease process is disseminate or focal. It has been my observation that disturbances of consciousness and motor function are the most constant symptoms of disseminate encephalitis. Early in the course of the disease, some affected animals appear excitable or restless; they may whine or bark more or less constantly. At first it is possible to make them stop by staying with them, or by talking to them, but later their noisy emotions cannot be controlled without sedatives. This period of excitement is of varying duration, or it may be so slight as to pass unnoticed. In the latter instance the first symptom noted is impairment of psychic reactions or dulness. The animals appear depressed, stuporous, or dumb; ordinary external or internal stimuli incite little reaction and they soon lapse into their former lethargic state. They may walk into a corner or against some object and remain there until forced to move. They do not resist being handled and palpation does not cause pain or discomfort.

Uncontrolled movements are frequent and the tendon reflexes usually are exaggerated. The latter may be unilateral or bilateral, and one or more limbs may be affected. Uncontrolled movements may consist of spasms of one or more groups of muscles, walking in a circle, rolling, rearing up on the hind legs, or suddenly bolting forward. The gait usually is staggering and clumsy, and the feet may be dragged or lifted unnaturally high. Affected animals not infrequently stumble when they are made to go up or down stairs or when they step over objects on the floor. They appear to suffer from muscular weakness, though this probably is due to their muscular incoördination and ataxia.

Affected animals generally will eat food if it is placed where they can reach it easily, although they may vomit it later. Epileptiform convulsions are an almost constant symptom but in some cases they do not occur until the terminal stages of the disease.

The pupils may be constricted or dilated. Frequently the iris reflexes are absent or uneven. Examination of the fundus may reveal retinal hemorrhages and papillary neuritis. However, in cases of chronic encephalitis in which there is marked edema of the brain, papillary edema may be present. Impairment of sight, hearing and smell are not infrequent.

The symptoms of focal encephalitis frequently are indistinguishable from those caused by intracranial tumors, and they are generally referable to definite portions of the brain. However, the most frequent symptom of focal encephalitis in dogs is the rhythmic clonic spasm commonly referred to as chorea or tic in dogs. This symptom is caused by a lesion in the basal ganglion.

An elevated temperature may be observed early in the course of encephalitis, but the temperature frequently is normal even though the symptoms are progressive.

Myelitis: Myelitis may cause pain and hyperesthesia, although the most frequent symptom noted is paralysis of both hind legs, or so-called posterior paralysis. The tendon reflexes are either normal or absent, there is no disturbance of consciousness, and vomiting and epileptiform convulsions do not occur. If the lumbar portion of the spinal cord is affected, fecal and urinary incontinence will result. If the ascending tracts of the spinal cord are affected, sensory disturbances will result, although these disturbances are not easily detected in a dog. Self-mutilation, however, has been observed because of peripheral anesthesia. If the dorsal columns are affected, the deep joint and muscle senses are abolished. An animal so affected may lift the feet unnaturally high in walking because it does not know their exact position. Ataxia and loss of equilibrium, when in the dark or when the eyes are blindfolded, have been observed in some animals with lesions of the spinal cord.

Pathologic Findings

Meningitis: The gross lesions of meningitis are not always clear-cut. However, usually one will note an increase in the cerebrospinal fluid, and not infrequently the spinal fluid is turbid or pink. The meninges may be hemorrhagic, but usually they

only appear thickened and a yellowish discoloration is not uncommon. The brain usually appears to be soaked with fluid, or wet, and the convolutions may be flattened. Hemorrhages around the vessels are not infrequent.

Microscopically, the pia generally appears thickened and is more closely adherent to the brain. Accumulations of leukocytes or lymphocytes, depending on the acuteness of the disease, may be noted in and underneath the meninges. The cerebrospinal fluid shows an increase in the number of cells present.

In chronic meningitis, deposits of calcium and bone may be noted in the meninges. This is especially marked in chronic pachymeningitis, a condition in which the dura is chiefly affected.

Encephalitis: The lesions of encephalitis usually are microscopic. Occasionally, however, hemorrhagic, softened, or fibrosed areas can be detected grossly. An increase in the quantity of cerebrospinal fluid is not infrequent. It may be clear and colorless, although it more frequently is turbid or pink. The brain often appears wet or edematous. This is more marked in cases of acute encephalitis.

The microscopic lesions vary markedly in their character, leukocytic and round-cell infiltrations, especially around the vessels, being perhaps the most common lesions. In the more acute cases the cells are chiefly leukocytes, whereas in chronic cases lymphocytes predominate. These accumulations of cells not infrequently so completely fill the lymph-space around the vessels that marked edema of the brain occurs. This causes the brain to appear vacuolated or spongelike microscopically. Degeneration and disappearance of the pyramidal cells and varying degrees of gliosis are noted. In my experience the lesions have been most marked in the cerebral cortex, in the region of the corpora striata and in the brain stem at the confluence of the cerebral peduncles. Focal lesions in the cerebellum in the region of the cerebellopontine angle were noted in one case. This caused symptoms typical of tumor in this region.

In many cases of encephalitis observed by me, degeneration of the pyramidal cells, resulting in their complete disappearance or in the formation of a halo or vacuole around them, was the only lesion revealed microscopically. This lesion, too, may be focal or diffuse. Cellular infiltration and gliosis were absent in these cases.

In some old cases of encephalitis following distemper in which tic or rhythmic clonic spasms of certain muscles are present, definite lesions cannot be demonstrated. These symptoms always

are due to a lesion in the cerebrum and some authors think that the lesion always is present in the basal ganglion. In cases in which the lesion cannot be demonstrated, it is possible that the symptoms are due to the destruction and complete disappearance of certain cells. If it were possible to count the cells before and after the occurrence of symptoms, a difference in the number of cells would be noted.

Myelitis: The lesions of myelitis may simulate those of encephalitis. They may be degenerative or atrophic in character, due to pressure from the outside or from within the spinal cord, or they may be caused by blood-vascular and nutritional disturbance. Inflammatory lesions in the spinal cord may be localized or disseminate. If the latter, they cause the condition termed "transverse myelitis." Perivascular cuffing and cellular infiltration are frequent. The cellular infiltration may be diffuse or palisaded. Hemorrhages, too, are frequent in acute myelitis. These may be macroscopic, but the cellular reactions always are microscopic in unstained sections of the spinal cord. In a case of hematomyelia observed by me, dilatation of the central canal was visible grossly.

I fully realize that the foregoing is a very brief discussion of meningitis, encephalitis and myelitis, but a comprehensive review of the subject would be too voluminous and burdensome to present as a discussion.

Illustrative Cases

The following ten cases of meningitis, encephalitis or myelitis are reported to illustrate the various types of symptoms and lesions observed in this disease in dogs. I realize that the discussion is very brief and reference to treatments has purposely been omitted.

Case 1: A German shepherd female, one year of age, underwent oöphorectomy on April 10, 1931. The operation was uneventful and she was taken home by her owner the same day. She remained in apparent good health until April 14, when it was observed that she appeared to be sick and refused to eat or play. Since this was the fourth day after the operation, the presence of generalized peritonitis was suspected. However, an examination revealed her abdomen to be neither tense nor sensitive and her temperature was only slightly elevated (102.5° F.). She was placed under observation without treatment.

On the following day, her temperature was the same. She still refused to move about and play, but she ate food when it

was placed before her. When she was forced to move, it was noted that she experienced difficulty in using her hind legs. She was very irritable, her pupils were unevenly dilated and the pupillary reflexes were practically abolished. Later during this day, she had several epileptiform convulsions and, following these, she did not recognize her master's voice or presence. She was given 5 grains of sodium barbital by mouth for its sedative action.

These symptoms remained essentially unchanged until April 18, when it was thought that some improvement was noted. She now could walk normally and ate her food, but she still failed to respond to spoken commands. This apparent improvement continued and, on April 21, she was definitely able to recognize her master by sound or smell although her sight was markedly impaired. Examination of the ocular fundi revealed bilateral retinitis and neuritis of the optic papilla. She was excitable and nervous. On the following day, marked exacerbation of all of the former symptoms was noted, and there also was slight rigidity of the neck and hyperesthesia. Her patellar reflexes were normal and there was no evidence of paralysis. She was destroyed by ether inhalation on April 24 and necropsy was performed.

Examination revealed her abdomen and thorax to be in perfect condition. When the cranium was opened, an increase in the quantity of the cerebrospinal fluid was noted. The spinal fluid was turbid and the brain appeared wet or edematous. Microscopic examination of the meninges and brain revealed clumps of leukocytes in the pia. The brain substance did not show any definite lesions, although all of the smaller vessels appeared engorged. A diagnosis of meningitis was made.

Case 2: A smooth-haired fox terrier, a male 14 months of age, was first observed by Dr. M. L. Morris, who shipped him to me for observation and diagnosis. The following clinical history was obtained: The first symptom noted was loss of memory. This was noted about one week before the dog was presented for examination. There was no history of a previous disease.

My examination over a period of two days revealed the following symptoms: The temperature was 101.0° F. The dog was unresponsive to ordinary commands or external stimuli, though he was not entirely deaf. His mouth remained partially open and the pupils were dilated and did not respond to light stimulation. Definite papillary neuritis was preesnt in each eye and marked impairment of vision was noted. The dog appeared

dumb; he would stay where he was placed on the floor until he was forced to move. When he walked, he traveled in a circle, counterclockwise, and his gait was staggering. He did not object to being handled by anyone, which is quite unusual for a fox terrier. Palpation of his body, head, or limbs did not cause pain or discomfort. His patellar reflexes were exaggerated and, when he sat down, his hind legs appeared spastic and would fail to bend. He manifested no definite paralysis or muscular weakness, but incoördination was marked. Cheyne-Stokes respiration was noted. Epileptiform convulsions, vomiting and urinary incontinence were not observed.

The diagnosis of a cerebral lesion was not difficult in this case, but its exact nature could not be determined until after necropsy was performed. Although epileptiform convulsions and vomiting had not been noted, internal hydrocephalus and the presence of an intracranial neoplasm had to be considered along with encephalitis. Necropsy revealed widespread edema of the brain, and this was confirmed by microscopic study. Cellular infiltration was marked in some areas (fig. 1). A diagnosis of encephalitis of unknown origin was made.

Case 3: A mongrel, male, wire-haired fox terrier, one year of age, was first observed by Dr. M. L. Morris. The dog had manifested mental disturbances, alternate excitement and depression. He had vomited occasionally, his appetite was poor, he urinated frequently, and his bowels were irregular. It was noted that he held his head tilted to the left, and walked almost constantly. Examination of the blood revealed leukopenia with a shift to the left. No filaria were present.

At the time of my examination the dog appeared excited and walked almost constantly. When standing still, his hind legs were spread as if he were bracing himself. Function of the right hind leg appeared to be impaired and the patellar reflexes in this leg were exaggerated. The pupils were dilated but contracted slightly when subjected to strong light. The ocular fundi were grossly normal, though the dog's sight appeared to be impaired. His head was held tilted to the left and definite disturbances of equilibrium were noted. Muscular rigidity or spasms and incontinence were not observed. A diagnosis of chronic focal encephalitis or tumor in the region of the cerebellopontine angle was considered. The dog was destroyed and a necropsy was performed. Gross lesions were absent. Microscopic examination of the brain revealed an area of degeneration, and gliosis was noted at the base of the cerebellum on the left

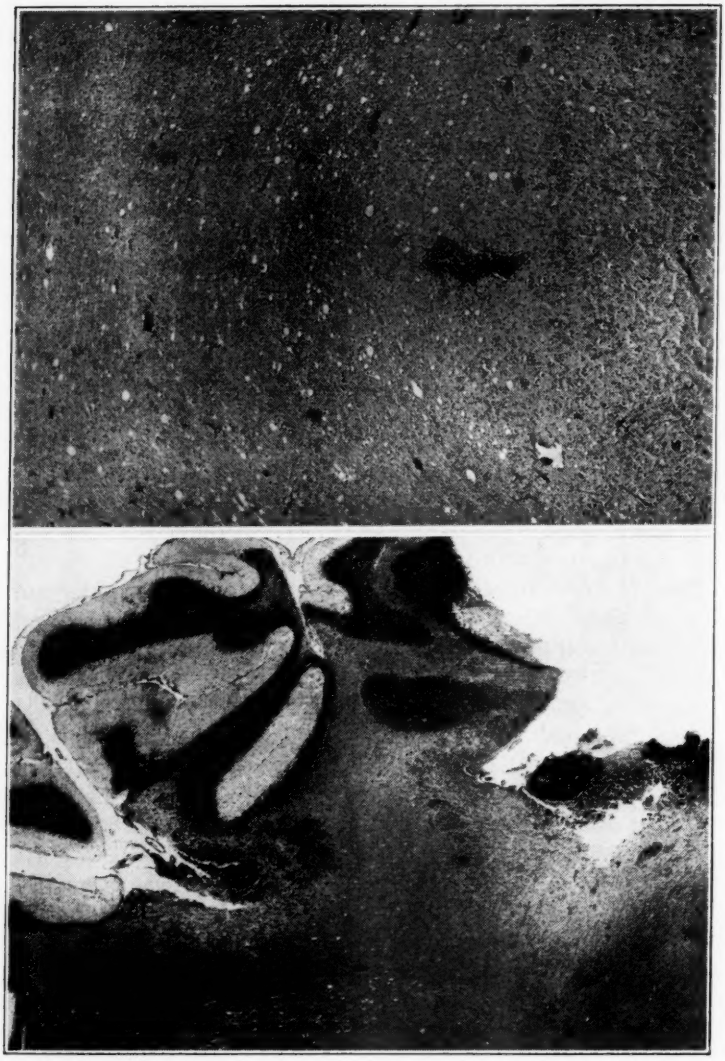

Fig. 1 (above). Marked, disseminate, perivascular cell infiltration. There is complete disappearance of the large nerve cells in this section. The vacuoles present may indicate the former site of the cells.

Fig. 2 (below). Showing the focal character of the lesions in the cerebellum. Degeneration of gliosis is well marked, indicating the chronic nature of the lesions.

side (fig. 2). This lesion appeared to be embolic in origin. There also were a few small areas of lymphocytic infiltration in the cerebral cortex.

Case 4: A Boston terrier, female, three years of age, was brought to me for observation and diagnosis on April 6, 1936. The following clinical history was obtained from the owner: About four weeks previously, it was noted that the dog was losing her sight. This condition progressed rapidly and she soon appeared to have lost her sight completely. Vertigo and ataxia had been noted, but vomiting or epileptiform convulsions were not observed.

On examination the temperature was 102.0° F. The dog was totally blind and pupillary reflexes were absent. When she was placed in a lateral recumbent position, she had great difficulty in arising. Vertigo and ataxia were marked, and she would fall when she was forced to turn sharply. When walking, the animal would lift all four feet high, a typical "goose-step." She was excitable, her respiratory rate was increased and her pulse rate was irregular. There was no noticeable impairment of hearing. Incontinence was not noted.

On the following day, the dog appeared to have some muscular weakness, and vertigo and ataxia were more marked. When forced to move, she walked counterclockwise and fell frequently. She refused to eat even when food was placed in her mouth. In the following two days, marked progression of the disease was noted. She was then destroyed with ether and necropsy was performed. There were no organic lesions in the thorax and abdomen. An increase in the quantity of the cerebrospinal fluid was noted on opening the cranium. The brain appeared wet or edematous. Microscopically many areas of cellular infiltration were observed in the cortex of the cerebrum, most marked in the posterior pole. Edema was marked (fig. 3). The accumulations of cells were chiefly lymphocytes. A diagnosis of diffuse, chronic encephalitis was made.

Case 5: A smooth-haired fox terrier, female, six months of age, was brought to me for observation and diagnosis on May 29, 1937. The owner furnished the following clinical history: The dog had been spayed about the middle of March; the exact date was not known but it was thought that her illness dated from the time of the operation. In the previous week, torticollis and epileptiform convulsions were noted. The head was pulled to the right and held there constantly. There was complete anorexia.

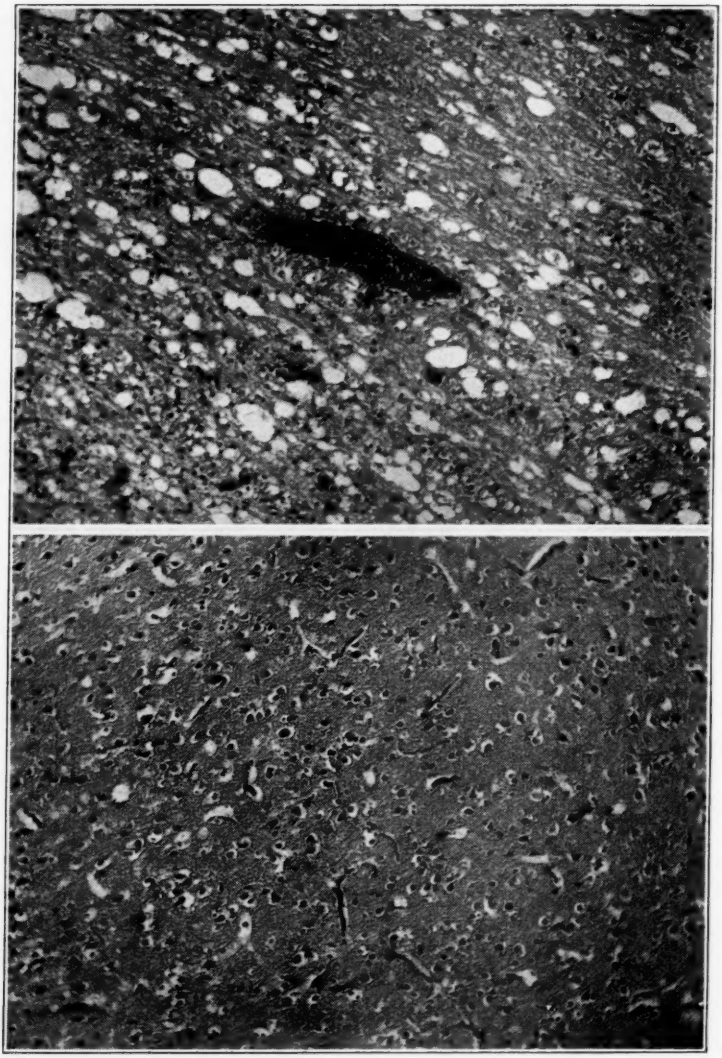

Fig. 3 (above). Chronic disseminate encephalitis, showing complete disappearance of the large nerve cells, perivascular cell infiltration and edema.

Fig. 4 (below). Showing each cell nucleus surrounded by a halo or vacuole. There is beginning degeneration of every large nerve cell present.

On examination, the temperature was found to be 101.2° F. The dog was in a fair state of nutrition. She could stand on all four legs, but she preferred to lie on her left side with her head drawn around to the right so that her muzzle rested on the loin. More or less continuous convulsive spasms affected her head, neck and lips. She also had repeated epileptiform convulsions while she was being observed. During these seizures she would draw her head up and backward and her left fore leg would be raised in a spastic extended movement. There was some spasticity of all four legs when she was lying on her side. The patellar reflexes were slightly increased. The pupils tended to remain slightly dilated, though the pupillary reflexes were not abolished. The optic fundi revealed bilateral retinitis and neuritis of the optic papilla. Disturbances of consciousness were present. The dog was mentally depressed and would not respond to commands, but she appeared to see or smell objects held before her.

Because of her seemingly hopeless condition, she was destroyed and necropsy was performed. The abdomen and thorax were in apparent good condition. When the cranium was opened, an increase in the quantity of the cerebrospinal fluid was noted. The fluid appeared cloudy. No other gross lesions were observed. Microscopic examination of the meninges and brain revealed areas of marked cellular destruction in the anterior and posterior poles of the cerebrum, most marked in the region of the corpora striata. There was one small area of cellular infiltration in the motor area of the anterior pole. The cerebral peduncles, cerebellum and medulla contained no definite lesions. The meninges appeared thickened and revealed some cellular infiltration in the pia. A diagnosis of meningo-encephalitis was made.

Case 6: A German shepherd, male, three years of age, according to his owner, had apparently been normal in the morning when he was permitted to run loose in the yard and on the street. He did not appear in the yard in the evening, so a search for him was instituted. He was not far from the house, but was both blind and deaf. Otherwise he appeared to be normal. It was thought that he might have been struck by an automobile or had gotten into a fight with another dog and thus become injured. On the following day, his condition was unchanged. His temperature was 101.2° F. His pupils were dilated and the pupillary reflexes were abolished. The fundi were grossly normal. He appeared to be mentally alert, though he was nervous and irritable. He trembled when standing, but manifested no

evidence of paralysis or weakness. His tendon reflexes were normal. Three weeks later, the dog was still blind and deaf. Disturbances of consciousness did not develop, but he appeared to have definite muscular weakness. No new symptoms were observed.

At the owner's request, this dog was destroyed and necropsy was performed. There were no demonstrable gross lesions in the thorax and abdomen. The cranium was opened and the meninges and brain were examined. The cerebrospinal fluid was not noticeably increased in quantity and was clear. The meninges appeared normal. Cross-section of the brain revealed petechial hemorrhages in the cerebrum. These were small, discrete and widely scattered. Microscopic examination revealed only small hemorrhages of varying size. There was no apparent cellular reaction around them and degeneration of the nerve cells was not noted. The optic nerves unfortunately were not examined. However, the presence of hemorrhages in the region of the fourth ventricle and in the corpora geniculata was thought to be sufficient to cause the blindness and absence of pupillary reflexes. A diagnosis of hemorrhagic encephalitis of indeterminate origin was made, probably caused by some toxic substance. An extremely high fever is known to cause petechial hemorrhages in the brain, but the weather was not hot at that time. Another similar case was seen by me the following year.

Case 7: A mongrel collie, female, approximately ten months of age, was immune to distemper and had suffered no severe disease previously. On January 29, 1937, she was observed to have a sudden, severe, epileptiform convulsion. Within three or four minutes, she partially regained consciousness, but she almost immediately had a second seizure. The convulsions then were almost continuous until an anesthetic dose of pentobarbital sodium was administered. The dog became rigid and cyanotic and attempted to vomit during each seizure.

Physical examination revealed marked swelling of the right hind leg. This evidently was the result of infection, as the leg was hot and appeared inflamed. Because of her relaxed unconscious condition following the administration of the pentobarbital sodium, further examination was not attempted. The following day, convulsions were not observed, but a definite change in the mental activity of the dog was noted. She appeared depressed and stuporous. Her reactions to ordinary external stimuli were practically absent. She refused to eat and preferred to lie quietly. Twitching of various groups of muscles was noted oc-

casionally. The swelling on the hind leg was beginning to suppurate. She became progressively worse during the afternoon and died that night.

Necropsy revealed the thorax and abdomen to be essentially normal. Examination of the meninges and brain revealed a marked increase in quantity and turbidity of the cerebrospinal fluid. The brain was soft and edematous and the vessels appeared engorged with blood. There were no gross lesions. Microscopic examination of the brain revealed marked hyperemia and congestion. All vessels and spaces were filled with blood and there were some small hemorrhagic areas. A few small clumps of leukocytes were noted in the pia. The most striking microscopic lesion was the marked destruction of the large pyramidal cells in the cortex of the cerebrum. In some areas all of these were partially destroyed, as manifested by a halo or large vacuole around the shrunken nucleus of the cell (fig. 4). A diagnosis of acute metastatic meningo-encephalitis was made.

Case 8: A smooth-haired fox terrier, male, three months of age, according to its owner, had had progressive weakness and loss of function of the hind legs for two weeks. He occasionally whined as if something were hurting him. Convulsions and vomiting were not observed. This dog was not known to have been sick or injured previously.

On examination, the temperature was 102.2° F. There was no definite mental impairment, but the dog did appear restless or worried. Marked weakness and partial motor paralysis of both hind legs were quite manifest. There also appeared to be some sensory disturbance in the hind legs and the patellar reflexes were diminished. The dog manifested no definite symptoms of pain on palpation of his head, body, or limbs, and there was no evidence of fecal or urinary incontinence. The pupillary reflexes were essentially normal and his sight appeared to be unimpaired. However, examination of the ocular fundi revealed areas of subretinal hemorrhage in the right and some edema of the optic papilla. His appetite was normal.

The disease progressed rapidly and, two days later, the dog was unable to use his legs. He remained conscious but became quite mentally depressed. The owner at this time wished the dog destroyed. This was accomplished by inhalation of chloroform, and necropsy was performed. Necropsy revealed no organic lesions in the thorax and abdomen. Examination of the brain revealed an apparent increase in the cerebrospinal fluid, which was clear and colorless. Cross-section of the brain showed some

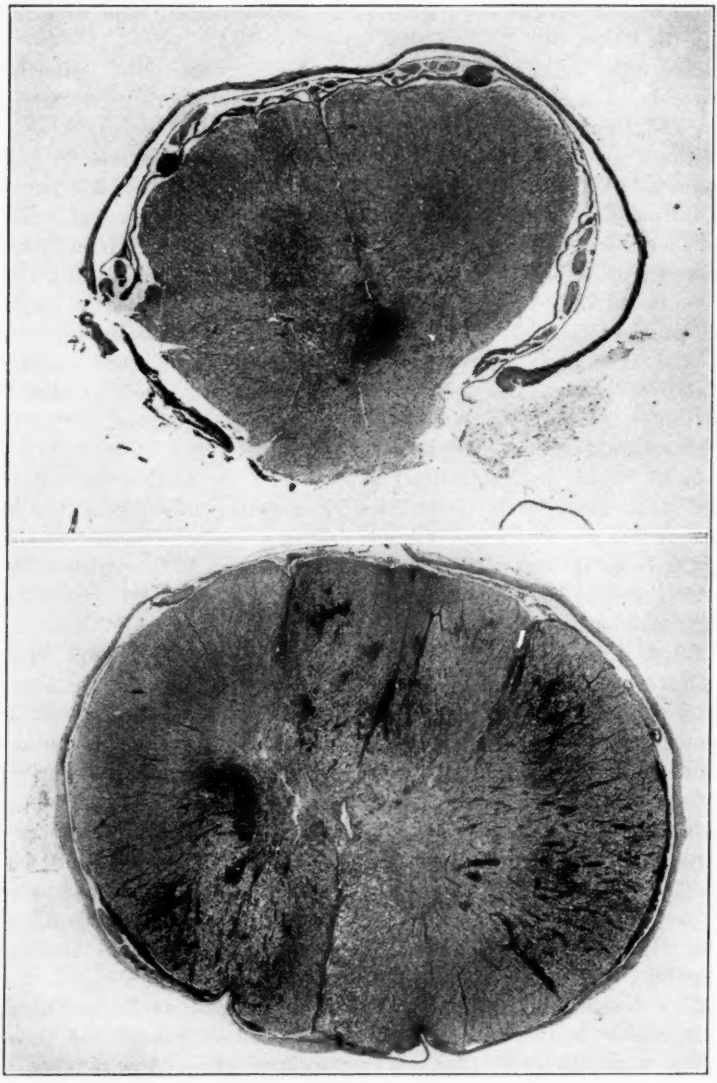

Fig. 5 (above). Hemorrhage in the anterior columns of the spinal cord. Other tracts and columns were affected at different levels of the cord.

Fig. 6 (below). Transverse myelitis, showing cellular infiltration and hemorrhage. All parts of the spinal cord are affected.

dilatation of the left lateral ventricle, and cross-section of the thoracic and lumbar spinal cord revealed several hemorrhagic areas (fig. 5). Microscopic examinations of the brain and spinal cord showed areas of lymphocytic infiltration in the spinal cord; the brain appeared normal. The cause of the hydrocephalus was not demonstrable. A diagnosis of myelitis and unilateral internal hydrocephalus was made.

Case 9: A springer spaniel, male, eight months of age, about four months earlier, according to its owner, had been given anti-distemper serum and, later, distemper vaccine. Following the second treatment, symptoms of encephalitis and descending myelitis developed. The latter was slowly progressive. The first symptoms noted were rhythmic clonic spasms of both fore legs. Some time later, progressive loss of function of the hind legs was noted, and flaccid paralysis of the hind legs and urinary incontinence then developed.

On physical examination, the sight, hearing, smell and psychic reactions of the dog were found to be apparently normal. The patellar reflexes were absent and the deep tendon reflexes in the forelegs were slightly exaggerated. There was anasthesia of the hind legs and perineum; at least, pricking the skin with a needle incited no reaction. The pupillary reflexes and ocular fundi were normal.

Because of the progressive nature of the disease and the existent motor paralysis, euthanasia was advised. This was done and necropsy was performed. There were no demonstrable lesions in the thorax or abdomen. The cranium and spinal cord were opened and the contents carefully noted before removal. There was a slight increase in the quantity of the cerebrospinal fluid, but it was clear and colorless. The meninges were grossly normal. The brain was removed and cross-sectioned. A moderate degree of internal hydrocephalus was noted. The brain substance appeared grossly normal. However, after it was fixed and sectioned and stained with hematoxylin and eosin, clumps of lymphocytes could be detected without the aid of a microscope. The spinal cord was also removed and cross-sectioned. It, too, was grossly normal, but after it was fixed, sectioned and stained with hematoxylin and eosin, some sections revealed such extensive cellular infiltration that they actually appeared blue. This was most marked in the lumbar portion of the spinal cord (fig. 6). Microscopic examination of the brain and spinal cord revealed the masses of cells to be chiefly lymphocytes. Definite cell inclusion bodies were noted in some cells. The significance of these

is not definitely known. A diagnosis of encephalomyelitis with moderate internal hydrocephalus was made. Symptoms of the latter lesion, however, were not manifested.

Case 10: A cocker spaniel, male, seven years of age, had had repeated attacks, about six months apart, of weakness of the hind legs, and posterior paresis since he was six years of age. At the time of the first attack, it was learned that he had a greatly enlarged and infected prostate gland. This was massaged, and he was given daily warm enemas and his diet reduced in quantity. On this regimen he recovered the motor function of his hind legs and was in sufficiently good condition to be permitted to go home after about a week of treatment. However, each subsequent attack was more resistant to treatment, and finally he failed to respond to any form of treatment at all. Paresis then progressed to complete paralysis of the hind legs and urinary incontinence was noted. Because of this the dog was destroyed and necropsy was performed.

Necropsy revealed the following lesions: The prostate gland weighed 17.5 gm, and histologically many infected areas and generalized cystadenomatous degeneration were noted. All other organs were grossly normal. The brain and spinal cord were removed for histologic study. Microscopic examination of the brain did not reveal any definite lesions. However, when the spinal cord was sectioned and studied, hematomyelia and marked degeneration of the central portion was noted in the lumbar and thoracic portions. The etiology of this is not known, but it is possible that it was caused by metastatic infection from the prostate gland.

The paralysis in this case undoubtedly was due to the lesion of the spinal cord. The condition was diagnosed hematomyelia.

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NON-PARASITIC SKIN DISEASES OF DOGS*

By S. W. HAIGLER, *Saint Louis, Mo.*

It will not be practical to attempt any detailed discussion of non-parasitic skin diseases in the alloted time of 20 minutes, and I shall therefore confine my brief remarks to those skin conditions that are most commonly encountered and are most baffling to the veterinary practitioner, namely, eczema and dermatitis.

The two terms are frequently used synonymously, some investigators using the term eczema almost exclusively, while others refer to the various cutaneous and subcutaneous skin reactions as dermatoses. To say the least, considerable confusion exists among veterinarians as to the proper nomenclature. Too much attention has been given to "what to apply and what not to apply to the skin" and too little consideration has been given to the etiology and proper handling of skin diseases. The terms eczema and dermatitis, as generally applied, are insufficient and vague and fail to designate, with any degree of definiteness, the common dermic affections encountered in small-animal practice.

In the light of the progress that has been made in human medicine, veterinarians are woefully deficient in the field of dermatology. I believe that we should attempt a definite, descriptive diagnosis in every case presented, and not be content with merely designating the case "eczema" or "dermatitis." The reason for this is twofold: First, a diagnosis of erythematous, vesicular, papular or pustular dermatitis immediately places the veterinarian on a more professional and scientific plane in the eyes of the client. Second, upon the simple diagnosis of eczema, the owner immediately thinks of the hundred-odd eczema cures that are so commonly and persistently advertised. Thus, when his dog again has eczema, you will likely find him procuring a bottle of some concoction at the corner drug-store and your repeat calls will be on the decrease.

Furthermore, inasmuch as this condition apparently is becoming more and more prevalent, the public is becoming more exacting in its demands and expectations of the veterinarian, and it is not only our duty but should be our sincere desire to meet this demand. A descriptive diagnosis, together with a complete history of individual case records of eczema and dermatitis, will also furnish valuable data from which a better classification

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might be obtained for our future guidance. How many of us have been guilty in the past of making a simple diagnosis of eczema in a case where there is a large, weeping, oozing area on the dog's back, and later an identical diagnosis where the lesions are entirely different? Suppose these two owners are neighbors and should meet on the street and discuss their veterinarian's diagnosis? Is there little wonder that the dog-breeder and kennel-man is called upon to treat some of our cases?

Etiology

In the classification of dermatoses from an etiological standpoint, a good deal of work has been done, but little in the way of definite results has been accomplished. While one investigator is proving that a large majority of cases are due to allergy or hypersensitivity, another is making equally convincing claims that they are due to metabolic and nutritional disturbances, and vitamin deficiencies, while still others are just as certain that the etiological factors are external in nature in the majority of cases. So, out of the confusing mass of claims and counter-claims, we are face to face with the realization that little or no definite knowledge is available as to the true etiology of these dermic reactions.

Milks[1] states that the question of defining eczemas was brought up at the International Congress of Dermatologists as late as 1930, and that Sutton, in his latest edition of "Diseases of the Skin," says:

> There is no satisfactory definition . . . it is only a sort of a dermatological scrap heap out of which, from time to time, certain diseases that present a characteristic and definite symptomatology are extracted.

Pomeroy[2] states that dermatologists are as diversified in their opinions as to the cause of eczema as they are in defining eczema and dermatitis, and summarizes several opinions as follows:

> Friedman divides the authorities into three groups: 1. The conservatives who believe that true eczema is purely endogenous in origin. 2. The radicals who state that eczema is merely a pathological process which can be produced by any number and variety of irritants of exogenous or endogenous origin. 3. The liberals who take a position between the two groups and exclude as rapidly as possible from eczema all forms of dermatitis that have definite causes, the removal of which brings about recovery.
>
> Highman states that eczema is a result of skin idiosyncrasy to precipitating causes, external in nature in the majority of instances and internal in the remainder.
>
> Stokes gives ten factors or components underlying eczema:
>
> 1. Hereditary or familial predisposition factor.
> 2. Dry skin factor.
> 3. Oily skin factor.

4. Pyogenic factor.
5. Mycotic or fungus infection.
6. Focal intoxication.
7. Metabolic factor—with special reference to carbohydrate metabolism.
8. Neurogenous factor.
9. Eczema, asthma, hay fever complex.
10. Allergy or hypersensitivity.

Now, when one considers the diversity of opinion of only these authors, is there little wonder that so much confusion exists with reference to skin diseases in canine practice? To these add other factors which, I believe, to say the least, are predisposing influences in dermic reactions, namely, breeding, feeding and environment.

In the breeding of dogs, man has ignored every principle of eugenics in his mad desire to produce individuals to his liking. I do not believe that anyone will question the fact that certain breeds are more susceptible to skin reactions than others, yet fanciers of these breeds have given little thought and exercised but few efforts to overcome the problem.

Much has been said about feeding, and much more remains to be said. Nutritional, vitamin and mineral deficiencies play a definite and distinct part in skin diseases. The trend of our modern civilization demands that "we reach for a can opener instead" or this might be termed to "reach for a box instead." In either event, the past decade has seen a tremendous growth of the commercial dog food industry and elaborate radio programs and newspaper advertising telling the public of wonderfully complete and balanced foods for the dog.

There are hundreds of different brands of dog food, and while some are very good, others have little nutritional value. I have seen case after case of dermatitis which was the result of continued feeding of certain brands of commercial dog food. Also, the same condition has been observed when unbalanced home feeding was practiced. In either case, a radical change in the diet will frequently result in complete disappearance of the disease.

Dogs, like men, possess idiosyncrasies to certain foods. Therefore, when a certain dog continually vomits or is otherwise upset by any given food, that particular food should be eliminated from the diet. In dealing with skin disorders, it is better to feed a diet that agrees with the animal, rather than with the disease.

Too frequent feeding and overfeeding are undoubtedly contributing factors to skin disorders. Every veterinarian is familiar with the early summer cases of acute eczema encoun-

tered in old house pets that are greatly overweight. Restriction of the diet, particularly the nitrogenous foods, is usually sufficient to overcome this condition.

Has the present-day environment of the city dog any relation to skin disorders? I believe that it has. The large dog that is confined to the efficiency apartment, or the heavy-coated animal that is kept in the heated home is certainly a "slap in the face" of Dame Nature. Also, do you believe that a promenade around the city block on a leash or a romp for five minutes in the vacant lot is sufficient exercise? When allowed its freedom, a dog is seldom seen walking, yet some people think that a short walk each day is all the exercise a dog needs, with no thought toward governing the diet accordingly.

I will merely mention such other factors as irritating antiseptics, soaps and oils; fleas, lice and ticks; fungus infections, and intestinal and blood parasites. Constipation, intestinal infections nephritis, and infectious diseases—which may be either primary or secondary—and there are still other factors.

Then what about the neurogenic patient—the highly nervous and excitable dog left in your care, while the owner is out of town for a few days, only to develop a nice oozing spot of moist eczema about the time he is due to go home? For a number of years, I have observed eczema of this nature in dogs. Just last month, in our practice, we noted a five per cent increase in these skin cases for the two weeks following the Fourth of July celebration, over the preceding two weeks.

The vitamin deficiency factor must not be overlooked. For the past year, we have been experimenting with a so-called vitamin-F supplement, and while no definite conclusions have been reached, there is every indication that vitamin-F deficiency is responsible for some cases of dermatitis.

Diagnosis and Treatment

Any sincere effort in the diagnosis and treatment of eczema or dermatitis should be preceded by a review of the anatomical and physiological functions of the skin itself. No attempt will be made at this time to review this field, as that information is readily available elsewhere. However, we must bear in mind that the skin is one of the organs of elimination, and any factor or factors which interfere with its normal function, whether they be external or internal, must be given consideration. Also, that the skin is a protective organ and as such is subjected to

numerous insults and injuries. Pusey explains the reaction of the skin as follows:

> When there is a balance between the capacity of resistance of the skin and the insults to which it is exposed, there is a healthy skin. This balance may, of course, be upset by variations of either factor to it; by exposure on the one hand, to insults beyond the capacity of the normal skin to resist (this he terms "dermatitis") or, on the other hand, by a lowered resistance of the skin so that it reacts from insults that the normal skin would bear without apparent damage (this termed "eczema").

There is a wide variance of opinion among veterinarians as to the choice of the term used. Some prefer to drop the term eczema entirely, using the more descriptive dermatological nomenclature such as erythematous dermatitis or pustular dermatitis, while others use the term eczema almost entirely. Several years ago, we almost discontinued the use of the term eczema in our practice, preferring the term dermatitis instead. However, the more I study the available literature, and the clinical cases encountered in practice, the more I am inclined to think that both terms may be used to advantage in veterinary practice.

If it were possible to offer in this paper a definite, clarifying, descriptive classification of non-parasitic skin diseases of dogs, I would feel that my efforts have not been in vain. However, I am frank to admit that a solution of this subject is too complex for me. There is much to be learned about skin diseases. We must continue to study, observe closely, and consider the complete history of the case, together with the evaluations of laboratory findings and perhaps by so doing we shall learn a little more about the proper diagnosis and treatment of these cases.

In considering the application of treatment, I believe that the methods employed are more important than the medicine used. One difficulty that confronts the veterinarian is the lack of opportunity to study his cases properly. You and I are expected to make a hurried examination and prescribe treatment that will effect a cure in a few days. On the other hand, it is not reasonable to expect the average dog-owner to be willing to spend a lot of money for this purpose, but he should be advised of the obscure nature of these skin ailments and not to expect too much.

The hair should be clipped closely well around the areas of moist eczema, and the entire dog clipped in cases of diffuse dermatitis. Clean the skin thoroughly with alcohol, hexylresorcinol, or some similar agent. If necessary to scrub with soap and water, I prefer to dry the dog thoroughly, and apply the above agents after the bath. In our experience, water seems to irritate and facilitate the spread of the infection. Local applications of

mild and soothing lotions, oils, ointments, or powders should be made as they may be indicated. If the animal is inclined to lick or scratch the infected areas, some method of restraint should be used or the area covered with bandage. A hypnotic dose of nembutal, chloretone, or some similar agent has proven an invaluable aid at the onset of these cases. In very acute cases of sudden onset, 0.5-cc doses of epinephrine hydrochloride subcutaneously every three to four hours for four doses have given remarkable results in some cases in our practice. Various arsenical preparations, fetal extracts, or other internal medication will prove valuable adjuncts in some cases.

With reference to internal medication, I am frank to admit that I do not know whether it does any good or not, but for a couple of years we have dispensed a preparation in the form of a brown tablet which contained, among other ingredients, five grains of sulfur. This tablet has been given right along in most cases of skin disorder.

After a good many years of dispensing of this tablet, I have noticed that there has been quite a brisk demand for more of these brown tablets. Upon inquiry the dog-owners told me that their dogs seemed to improve rapidly when given the tablet. Perhaps, six months later, the skin would break out again. They would repeat the dosage and the eruption would clear up.

During the course of the year, some high-powered salesman came along and sold me another tablet which happened to have a red color. It was a good tablet, but it did not contain sulfur. We dispensed that tablet along the same line, but frequently people would come back with this statement:

"I want some more of the brown tablets. The red tablets don't do any good."

Now, I am wondering just what effect the old remedy of sulfur and molasses may have had in that case. Perhaps sulfur and molasses has more virtue than we give it credit for having. I believe Dr. Reihart told me that the sulfur metabolism varies considerably in various breeds of dogs, being lowest in the Boston terrier group, and highest in the larger breeds of animals.

From my experience I am sure that we have more cases of skin troubles, both parasitic and non-parasitic, in the terrier group. So, perhaps, after all, there might be some virtue in some internal medication.

An addendum to a paper presented at the Eastern States Veterinary Conference in New York City last month by Dr. E. B. Dibbell, of Baltimore, contains some 28-odd favorite formulas

used by veterinarians all over the United States. Not only does it contain valuable information, but it is interesting to note the different formulas used to accomplish a common purpose.

In conclusion I would like to repeat that there is a growing demand and a long-felt need for a better definition and classification of non-parasitic skin diseases. We can well follow the progress of human dermatologists in this field, but must ever bear in mind that we are dealing with canine patients, and that our problems differ somewhat from the physician. Ours is a veterinary problem and must be solved by the veterinarian. Perhaps a little more careful observation and study, together with complete records of every case encountered, will eventually help us to solve the puzzle.

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Discussion

Dr. T. J. Leasure: I wish it were possible for the veterinary profession as a whole to adopt a more uniform classification and nomenclature for covering dermatitis as a skin disease. Dr. Haigler covered his topic very thoroughly and I do not believe I can add a thing to it. (Applause)

Dr. Henry H. Baur: Is that five-grain sulfur tablet a put-up tablet, or do you make it yourself?

Dr. Haigler: It is merely a proprietary preparation. This particular tablet is made by a firm in Pittsburgh.

Dr. O. Norling-Christiansen: I should like, first of all, to commend Dr. Haigler on his excellent paper. We have a great problem in skin diseases. One comes to my mind, however, that I do not believe Dr. Haigler mentioned. In many cases, dermatitis will come from disorders of the anal glands, anything from slight disorders to complete abcessation.

In our hospitals we have seen, perhaps, hundreds of cases where the evacuation of these glands, or operation in a case of abcessation, has completely restored the animal's condition back to normalcy, with no other treatment following.

Another thing, which we use in many cases as a regular routine, is a complete gastro-intestinal treatment when the animal is in physical condition to stand it. I believe that that has a great bearing on some of the conditions. However, we all live to learn, and there is plenty to learn about skin diseases in dogs.

Dr. Haigler: In 1932 or 1933 I heard a statement made in a paper presented in Kansas City which said that 99 per cent of the cases of dermatitis on the backs of dogs were caused from anal gland infection. I thought that was quite a remarkable statement at the time. Frankly, I have observed this condition quite closely since that time. I have seen this same condition occur in dogs whose anal glands, as far as I knew, were perfectly normal in their functions. I have seen the same condition in dogs whose anal glands were abscessed. I have removed the anal glands completely and have still had the same condition occur, so I am unable to say whether it is due specifically to anal gland conditions. It is, perhaps, a factor.

DR. CHRISTIANSEN: I believe Dr. Haigler misunderstood me. I had no intention of saying that dermatitis in any form was specifically caused by anal gland trouble. I agree thoroughly with Dr. Haigler that we see it in normal dogs when there is no disturbance of the anal glands, and also in dogs where there is a disturbance. I said "several hundred cases," and that is not very many over a period of five years. There are cases where definitely the condition can be cleared up by treating the anal glands.

MEMBER: I should like to ask Dr. Haigler if he has noted any difference regarding atmospheric conditions. In 1934, it was rather dry throughout the Central West and rather hot. In 1935 it was rather wet. We had considerable moisture. The humidity was great. In 1936 it was dry and hot again, and this year we have a relative high humidity.

In our own practice we notice a considerable connection between this so-called eczema, or dermatitis, and relatively high humidity. Do you notice any difference between 1934 and 1935, and 1936 and 1937? When the weather is dry and hot, with no humidity, it seems that there is an evaporation of the excretions and there is no skin irritation. Have you noticed that atmospheric conditions have any bearing on this?

DR. HAIGLER: Perhaps there is a difference. There are more cases in the summer than at any other time of the year. But, frankly, I do not know. However, I do know that we have eczema and dermatitis from Maine to California, and all over the world. There are various kinds of dermatitis. We have it in health resorts, where the weather is of one condition, and we also have it in the North and the South, where the weather is completely different. We do find more cases in the hot months than at any other time of the year.

Data on Cancer in Animals Wanted

TO THE EDITOR:

In connection with some research work now being carried out at the Memorial Hospital for the Treatment of Cancer and Allied Diseases of New York, I am desirous of obtaining as many verified instances as possible of cancer of the oral cavity in animals.

I shall be deeply grateful to any veterinarian who sends me any case reports of intraoral cancer, giving the species of animal and as much specific data as possible as regards the exact location, the size and the clinical history. I particularly wish the loan of the histologic slide upon which the diagnosis was made, and will return any such slide, if the sender so wishes.

Please address all reports to Dr. Hayes E. Martin, at 30 East 72nd Street, New York, N. Y.

HAYES E. MARTIN, M. D.

New York City, March 25, 1938.

Diamond Jubilee Convention
Hotel Pennsylvania—New York
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THE CONTROL OF THE MILK SUPPLY FOR AN IOWA INSTITUTION*

By W. A. RISER, *Glenwood, Iowa*

The dairy industry in Iowa has grown until now it ranks in importance with that of any state in the nation. Until recent years, Iowa was considered a corn and hog state and not a dairy state. Now, however, it is recognized as one of the leaders in the dairy field. This being true, the Iowa veterinarian must take the dairy occupation more seriously. The veterinary profession seems, however, to have been a little slow in grasping the importance and in sensing the needs of this industry. If there is one agricultural pursuit in which advice and supervision in sanitation is needed for continued existence and progress, that is the dairy industry.

The statement has been truly made that more men have failed in the dairy enterprise because of their inability to control disease than in any other line of animal production. The profession is overlooking an apportunity to render a service if it does not equip itself to meet the needs of the dairyman by advising in disease control and sanitation. The dairyman needs this advice and solicits it. If the veterinarian is not qualified to meet this request, his client is going elsewhere for help.

The best time to talk sanitation to a dairyman is when he is in trouble. Infection is so prevalent among cows that, sooner or later, the dairyman will present his problem to the veterinarian for consideration. This is the opportunity to spend some time in talking over the matter with this man, convincing him that his present plan is wrong and offering him a better one. It should be the aim of the veterinarian to sell the program so well that he will secure the complete confidence of his client. He must likewise obtain the promise of the owner that he will adhere to the program regardless of whether improvement is immediately accomplished or not, because in badly infected herds often six or more months pass before results are obtained. This must carefully be explained to the dairyman so that he will not discontinue the program before it has a chance to speak for itself.

I have had the pleasure of working with the dairy herd at the State Institution for Feeble Minded Children at Glenwood, which has made a remarkable record in disease control and sanitary management since the adoption of a rigid plan 4½ years ago.

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

Survey

A careful survey of the dairy was made to determine some of the factors which might be responsible for a poor quality of milk and low production. The herd was tested for Bang's disease; the cows were examined for pregnancy, the bulls tested for breeding potency; the cows were examined for breeding efficiency and production ability. The records of the calves were studied for liveability and maturity. The equipment was inspected for cleanliness, repair and care. The milk was examined for dirt particles and number of bacteria per cubic centimeter. The findings from such a survey were as follows: 30 per cent Bang infection, 25 sterile cows, 50 per cent mortality in baby calves, equipment in bad repair and bacteria counts exceedingly high.

Herd Records

The first essential of herd management is herd records. This point cannot be stressed too strongly. By herd records I do not mean just production records. I mean a complete record of each individual animal from the day its dam and sire were mated until it is retired from the herd. Such a record should contain a complete pedigree of the dam and sire, including production and performance data, mating date of dam and sire, date of birth, Bang's disease agglutinatior titre of dam, and peculiar incidents that may have happened during pregnancy. If a female calf is born twin to a male, special note should be made. When the animal becomes older, its record should contain such things as breeding dates, pregnancy examination dates, freshening dates, production records, agglutination records, udder examination records, hospital records, and symptoms shown and treatments rendered.

To keep a complete record as outlined above is a big job, but when trouble is encountered with a particular animal such a record is very valuable. This record is absolutely necessary to treat any dairy animal intelligently and scientifically. I have some blank record sheets with me, and I would be glad to explain how they work if anyone is interested in them. Complete records must be kept if success is to be obtained in herd management.

Calf Mortality

The best asset to any herd is its young animals that are successfully raised. A herd would very soon go out of existence if it were not for the heifers growing into production. Since

Utah State Agricultural College

saving calves was so important and since this herd had such a high mortality in baby calves, immediate attention was given to improving this department. Investigation of the care and feeding of calves was made to determine its influence on the exceptionally high death loss. Postmortems were held on all calves succumbing to infection and bacterial cultures made from the pathological tissue. The conclusions drawn from this procedure were that the calves were suffering from pneumonia, scours and navel-ill. Pneumonia and scours were the most common. It was also concluded that incorrect feeding, poor ventilation, lack of sanitation, and exposure were contributing factors to the above diseases.

A program to correct the contributing factors and to control the disease by the use of vaccines was adopted. The routine use of vaccines at the time of birth was established. This produced considerable shock in these baby calves. Their resistance was greatly lowered, they developed symptoms of infection and died in a shorter period of time than they did when no vaccine was used. The degree of shock was more or less constant with the use of any of the broth culture or bacterial suspension vaccine or antiserums. Little result was obtained from vaccines so it was agreed to stop using them. We do not mean by this that vaccines are of no value in calf diseases, but we are merely stating that vaccines did not work in this particular herd.

The system of calf-raising adopted began with the cow before the calf was born. Good management requires that the cow be dry six to eight weeks before freshening. This gives her sufficient time, if fed an adequate ration, to rebuild her body reserves and meet the demands of developing a fetus strong and mature at birth. A few days before calving, the cow is taken from the dry-cow pasture and placed in a maternity stall which has previously been scrubbed with hot lye water and well bedded with clean straw. (Formula of lye water, as mentioned in this paper, is one can of lye to 15 gallons of hot water.) A few hours before the calf is born, the tail, udder, anus and vulva are thoroughly scrubbed with soap and water. The udder is again washed with soap and water after the birth of the calf and before it is allowed to nurse its mother.

The calf is left with the mother 12 to 18 hours before it is taken to the calf barn, where it is placed in a freshly disinfected stall. During the first four days of the calf's life, it receives its mother's milk because the first milk of the mother is high in colostrum. This complex protein is said to contain antibodies

and body regulators which make the new-born calf more resistant to disease organisms.

Feeding the Calves

When calves are hand-fed, certain well-established principles must be carried out to avoid digestive disturbances. The baby calves are fed three times daily from milk produced from negative cows with healthy udders. The containers used for feeding are thoroughly sterilized and the amount of milk for the individual calf is not to exceed three-fourths of a pound daily per each 10 pounds of live weight; to this is added one-half pint of limewater three times daily. One or two drams of cod-liver oil is added to the calf's milk in winter weather. If the daily milk consumption is increased beyond this amount, the delicate system of the baby calf is very susceptible to intestinal disorders. A grain and hay ration is added to the diet after the first ten days.

Muzzles were placed on the calves to prevent them from ingesting infected material, such as straw and infected feces, but they did not prove very satisfactory because of trouble encountered in keeping them properly adjusted. They also caused blisters to form on the heads of the calves.

The above sanitary precautions and revised methods of feeding did considerable to correct the high mortality but the death rate was still too great, so new quarters were sought for them. A new barn was built, equipped with individual stalls in which the calves were isolated for the first 30 days and then they were kept in small groups. When the calves were 90 days old, they were returned to the original calf barn because they had developed enough resistance to the infections at this age to prevent further trouble. A univent steam heater was installed in the new calf barn to prevent temperature changes. When the above plan was perfected, the calf troubles for the most part were solved. The success in raising a well-matured, healthy calf depends upon absolute adherence to the most minute detail of a well-developed and organized plan. Therefore, the most responsible employé of the dairy must be designated to carry out this well-outlined program.

Bang's Disease

The presence of Bang's disease reactors in a fine herd of cattle is always discouraging and when a herd is known to contain 30 per cent infection, it is a real problem to decide just what should be done with it. Equipment was not available for segregation of the infected animals at the time this work was started.

Slaughtering the animals was prohibitive because of the great demand for milk in the institution and because some of the cows were needed to produce calves for future additions to the herd. The reactors were segregated in the lower end of the barn. They were milked last, grazed in an isolated pasture, and watered in separate tanks. Testing of negative animals was continued at ninety-day intervals.

This method was found to reduce the spread of the disease considerably, but not entirely. Soon afterwards, an isolation barn was built, the reactors moved into it, and as soon as they gave birth to calves and went out of production, they were slaughtered. When the segregation barn was established, the negative herd was tested at six-week intervals. There were less reactors at each test and after a period of six months the herd gave a clean test. When Bang's disease was discovered in the herd, the milk from positive cows was poured in special containers and used for cooking.

Other Diseases

The breeding efficiency of this herd was very low as shown by the survey of the herd. Bang's disease infection was partly responsible for a number of cows that were hopelessly sterile. Others were discharging pus, due to metritis or cervicitis.

These cases as presented required a great deal of treatment, patience and observation before results were obtained. Cows discharging pus are always sources of danger to other cows. The cows so affected were isolated and not permitted to be bred until free from infection. Bulls often acquire and pass this pus infection from cow to cow. It is wise to have a designated bull to which females that have harbored this infection may be bred. No cows should ever be bred following treatment until they are first examined with a speculum for pus and found to be free. Breeding of cows that are discharging pus aggravates their condition, delays recovery, and exposes the bull to possible infection.

The cow that is the most valuable to the herd is the one that produces a good flow of milk and gives birth to a live healthy calf at least once every twelve months. One of the chief aids in such a program is an accurate account of the age and history of each cow. The essential things that mark for regular breeding in a herd are accurate breeding records, isolation of cows at calving time until the membranes come away and the vagina is free from discharge.

Cows Should Be Rested

Producing milk and furnishing food for a growing fetus in the uterus is a real job for a cow. Therefore, she deserves a sixty- to ninety-day rest before she is bred. At the end of this period, the cow may be bred providing she is clear from purulent discharge. If she fails to come in heat again when she is bred, she is examined at six weeks after breeding. If pronounced pregnant, she is not bothered again. If she comes in heat again, she is bred a second time. If she fails to conceive this time, she is examined, the trouble diagnosed, and the condition treated.

The entire program of sanitation points to one primary goal—that of the production of a clean, safe, wholesome product. The superintendent's interest in the dairy lay in finding methods of producing a better product for his children. It is his job to see that his 2,000 children are fed, housed and clothed in the best available manner. Therefore, he demands that each item that goes for the children's well-being is above reproach.

A dairy product, to fulfill such a requirement, is dependent upon a vast scope of elements, if its safety is to be beyond doubt. The departments responsible for the wholesomeness of any dairy product fall under three heads—the employés, the animals, and the equipment. Any one of these three departments may render the product unsafe, and the product, on the other hand, can not be safe without the highest degree of coöperation between all three departments.

Health Examinations of Employés

Milk can be the safest, most wholesome, most healthful food to mankind, or it may be the reverse. Milk is an ideal medium for the growth and transmission of a great number of severe infections of man. A great many times, man is harboring a chronic infection and milk acts as a carrier for this infection to the consumer of the product. Therefore, it is always necessary to examine all employés at regular intervals to be sure they are not carriers of chronic infections. Everyone connected with the dairy at this institution is subjected to a rigid examination by a staff physician at certain specified intervals. Such examinations consist of observations for throat, nose, sinus and skin symptoms along with an x-ray plate of the chest. This procedure, with a complete family history of health conditions, usually eliminates the possibility of an employé being a source of contamination of the milk. If an epidemic of sore throat, scarlet fever, or pox is experienced, a check is made on all dairy em-

ployés for cases of corresponding infections. The udders of the cows are also given a rigid physical examination for mastitis.

When this work was started, the herd contained several cases of mastitis and a few new cases have developed since that time, but we have never encountered an acute outbreak. Early detection of mastitis is essential to safeguard the milk from contamination. This is accomplished by the use of the strip-cup on each cow before each milking and the periodic physical examination of the udder by the veterinarian.

Care of Milking Equipment

Low bacteria counts can never be maintained if milking equipment is not clean and sterile. The technic employed for the care of this equipment is as follows: When milking is completed, the utensils are washed with cold water to remove the milk, then scrubbed with an alkaline washing powder, then rinsed with hot water and placed in the sterilizer until the next milking. The working parts of the machine are washed with large quantities of cold water to remove the milk; then live steam is run through the system for 15 minutes. The steam is then blown out of the system and it is left until the next milking. Once a week, the entire system is taken down and thoroughly scrubbed. Just before the next milking, chlorine solution is run through the system, which is then rinsed with tap water. The teat-cup units are treated in the same manner, except that they are allowed to stand in lye solution between milkings. Lye solution is made by adding a teaspoonful of lye to one gallon of sterile water.

Within the last few months, a milking parlor has been installed and used for milking all healthy cows. It is a six-unit system and milks 48 cows per hour. The cows are previously groomed, the hind quarters clipped, and the udders washed before the cows enter the parlor. Before the cow is milked, the udder is tested with the strip-cup. Each cow is milked in an individual compartment in the parlor and then returned to her stall. The teat-cup units are rinsed with cold water and sterilized with live steam before being placed on the next cow. The milk obtained from the cow being milked goes into a receptacle where it is weighed and withdrawn to the aerator by suction. Sterilized milk-cans are then filled with this milk and placed under refrigeration at 25° F. ready for delivery to the specified kitchens.

Results

Some might say that this effort and the expense are not necessary, but let us compare the survey taken in March, 1932, with

a survey taken January 1, 1937, to see if results have been obtained.

Milk production was raised from 269 pounds of milk per cow to 429 pounds per cow. Bacteria counts were lower than grade A milk. The mortality of baby calves was lowered to 5 per cent. Three cows are being treated for sterility and the rest of the cows are either pregnant or not ready to be bred.

Conclusion

In conclusion, the one thing that must be observed above all else in dairying is a strict adherence to the principles of sanitation, no matter what specific plan of control is adopted. Vaccines and drugs are not cures for infectious diseases in a dairy —they are only aids. A clean healthy herd must be maintained before production and economy can be assured. Neglect of sanitary measures in order to reduce expense is false economy and the herd will soon suffer, and the efforts of the dairyman are doomed to failure and disappointment.

Whitney Veterinary Club

The Whitney Veterinary Club is somewhat out of the ordinary as veterinary organizations go. It was formed in January, 1935, at Jonesville, Mich., by a number of veterinarians who got together to honor Dr. J. C. Whitney, of Hillsdale, Mich., on the occasion of the 50th anniversary of his graduation from the Ontario Veterinary College.

Ever since its organization, the Whitney Veterinary Club has met once a month with very few misses. The organization has no officers but this has been no handicap. The club meets at the home of one of the members each month and the host of each meeting acts as the chairman of the program committee. Questions of vital interest to the veterinary profession are discussed by the members each month, while their wives enjoy a social evening with the hostess.

The territory covered by the membership of the club extends from Sturgis, Mich., on the west, to Monroe, Mich., on the east, and from Lansing, Mich., on the north, to Wauseon, Ohio, on the south.

Diamond Jubilee Convention
Hotel Pennsylvania—New York
July 5-6-7-8-9, 1938

CONTROLLING POULTRY DISEASES*

By F. C. TUCKER, *Claypool, Ind.*

We do not need to go very far in the past to recall the time when it was unnecessary for the veterinarian to possess any knowledge of poultry husbandry or of poultry diseases. But the high development and wide distribution of the poultry industry in recent years has created a new field of service for the veterinarian. Today, poultry is one of the foremost industries of agriculture, is highly specialized and requires accurate and scientific knowledge. The industry is here to stay. Moreover, the principles of health and disease are no different in poultry than in any other live stock industry. When fundamental facts are overlooked and principles of sanitation are neglected, many disease problems will be encountered. Many millions of dollars are invested in this major agricultural industry and its income is exceeded by only two others, the dairy industry and swine production. In the conserving of invested capital and the protection of income, the veterinarian has a very important part to play.

In recent years, the veterinarian has shown a changed attitude toward poultry practice. Formerly he seemed content to permit the itinerant or quack to monopolize the field of disease control. We of the older practitioners can vividly recall that the same conditions existed in regards to swine practice. Because of the wide distribution of the industry, it is my opinion that a very large percentage of the country practitioners have the opportunity to render service to many poultry clients. The veterinarian has a very important rôle to play in the production of healthy, liveable baby chicks. A thorough knowledge of poultry diseases and poultry husbandry will enable the veterinarian to render service that will be highly appreciated.

To be in a position to render efficient service, it is necessary that we fortify ourselves with sufficient knowledge of poultry diseases, disease control and sanitation, or almost any problem pertaining to poultry husbandry, so that we can talk intelligently and convincingly on those subjects when poultrymen come to us with their problems. If we do not have sufficient knowledge to talk intelligently concerning their poultry problems, it is most likely we will experience some difficulty in "selling ourselves" to them. And our remuneration will be in proportion to our success, provided our relations are carried out in a business-like manner.

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

The modern veterinarian of today is a man trained to view his work as a genuine servant to the live stock industry which, in most localities, includes service to the poultrymen. One of the most important diseases affecting poultry, one of the most fatal and most destructive is pullorum disease. The control of this disease affords a veterinarian an opportunity to render a service to his community that is highly appreciated. When the hatcheryman and veterinarian coöperate in their efforts to control this disease, having an intelligent understanding of its nature and the natural methods of transmission, the results of their efforts will most likely be entirely satisfactory.

Lack of satisfactory results usually can be traced to some "leak" in the program, such as the addition of new blood of unknown health status, custom hatching, or feeding infertile eggs. "Short interval" retesting until the reactors are reduced to at least 5 per cent will assure liveability of baby chicks. It is highly gratifying to the hatcheryman to receive reports of high liveability from his chick-buying customers, and in turn it reflects credit upon the veterinarian who has the responsibility of conducting the tests. Many hatcherymen have the interest of their chick-buying customers at heart and will leave nothing undone in their efforts to produce a baby chick of high quality in which liveability is assured under reasonable brooding conditions.

Times Have Changed

Time was when the hatcheryman and feed-store-man "passed the buck" to each other as to the causes for chick losses. One common way for the hatcheryman was to visit the chicks, post some and show pulmonary congestion, and then tell the owner that he chilled the chicks on the way home from the hatchery and that they are dying from pneumonia. I have known hatcherymen to state that week-old chicks were dying from coccidiosis. And in all this confusion and high-pressure salesmanship, the innocent buyer paid the bills. Conditions are quite different now, as old John Q. Public knows something about this disease himself. He knows that when he has a heavy death-loss in his brood of chicks during the first two or three weeks of their life, there is something wrong with the hatching program and it is not hard for him to figure out when his brooding conditions are the same as that of his neighbor whose death-loss is reduced to the minimum.

When a brood of chicks is bought in the spring the purchaser really is after a flock of good laying pullets in the fall. The

price paid, whether high or low, does not determine the cost of these pullets when they are housed. A number of other factors must be considered before the true cost can be determined and all factors center around quality. Therefore, good quality of the chicks themselves is more important than the price and should include the ability to live and grow into pullets of uniform appearance and good breed characteristics, and with large frames and good body weight.

Pullorum Disease

In our efforts to control pullorum disease let us not lose sight of the fact that unfortunately all of the infected chicks do not die from the disease in chickhood, for as many as 25 to 50 per cent of the infected chicks may survive the attack and become carriers. It is these surviving ones that must receive our earnest attention when the next crop of baby chicks is considered. The organism, *Salmonella purllorum,* will live in the blood-stream of the baby chick, later on localize, most commonly in the reproductive organs, and remain as a focal infection. The only way to control this disease successfully is to prevent the eggs from these birds that survive the attack in chickhood from getting into the incubators. If one is interested in controlling this chick disease, it is well for him first to consider the different tests used. The value of the best selected should be determined by its known ability to identify this "carrier" hen.

Before any attempt was made to do testing, I first consulted the Veterinary Department of Purdue University as to the efficiency of the different tests being used and they were not hesitant in telling me that, if I really wanted to control the so-called B. W. D. (Bacillary white diarrhea) in baby chicks, the tube agglutination method was the one to use. I then procured equipment for that method of testing, tested 2,000 breeders for a hatcheryman whose death-loss had been such that he had decided to quit the hatchery business if it was not possible for him to put out a better grade of baby chicks. His experience with pullorum disease was such that when he saw a customer returning to his hatchery in ten days or two weeks after buying his chicks, he was sure of receiving more complaints on live-ability. What he wanted to do was to produce a better grade of chicks—healthy liveable chicks—that would grow and develop into an even, healthy flock of pullets. This hatcheryman was "conscientiously disease-eradication-minded."

It is with this type of hatcherymen that veterinarians can make rapid, forward strides in the control of this disease. After one

year of testing, this hatcheryman was thoroughly convinced as to the merits of the test. The second year he was not backward in advertising blood-tested baby chicks. In fact, for a few years, his advertising contained this phrase:

BLOOD TESTING IS THE SAME TO THE POULTRY INDUSTRY AS SPRAYING IS TO THE FRUIT-GROWER

That phrase is original with him. This hatcheryman is still very enthusiastic for the test. He was the first in our section of the country to guarantee 95 per cent liveability of the chicks for two weeks, a practice he still continues and regards it the best advertising he can do. He has a form he fills out for the purchasers when they get their chicks which specifies that if they return after 14 days with a greater death-loss than 5 per cent, he promptly repays them in cash, not in chicks of a different age or at half-price, but the purchase price in cash. He and his hatchery have a reputation for quality products and square dealing. In our community he is a leader and an authority on poultry. His enthusiasm and support have been a great influence in the progress made in pullorum disease control in Indiana.

INTRADERMAL TEST DISCARDED

There is considerable work attached to this method of testing and it involves quite an outlay for laboratory equipment. I had not been engaged in poultry practice very long when I thought the intradermal or wattle test was receiving considerable support from those who, I suppposed, were authorities to judge its merits. I used that test on some reactors and thought my results were satisfactory. I looked forward to the next year's testing with that method, which would mean quite a saving in time, labor and equipment. About that time, I consulted one who is nationally recognized as an authority as to the value of the intradermal test in detecting the "carrier" hen and his advice was that if I wanted to stay in poultry practice to let the wattle test alone. I took him at his word. I have found that the tube-agglutination method of conducting the test is a very reliable and dependable agent to employ in the control of pullorum disease and if the veterinarian and hatcheryman are really interested in producing a high quality chick, they can each be a potent factor and a worth-while servant in their community and especially to the chick-buying public in their territory through this method.

I have had a very limited experience with tests other than the tube test. However, I have some acquaintance with results others

are having with different methods of testing. One flock of New Hampshire Reds with which I am well acquainted has the following history: They were bought five years ago, directly from one of the larger breeding farms in the East, and were the first pure-bred New Hampshire Reds in our community. I tested that flock of birds the next breeding season, with a little less than 5 per cent reactors, and subsequently they were tested each year, by the owner with the whole-blood method. A year ago, some difficulty was experienced with liveability of baby chicks, so this year the flock was tested twice with that test and had about 3 per cent reactors each time. When chicks were sold this year from that flock, the death-loss was greater than before and 20 to 30 per cent mortality was not uncommon, some losses running as high as 50 to 75 per cent.

In former years, I had done considerable testing for this hatcheryman and we were still good friends. He knew the merits of the tube test and I was again engaged to test the flock of about 400 birds, which showed nearly a 20 per cent reaction. Results following the elimination of the reactors were entirely satisfactory and no more complaints were made on liveability during the season. A laboratory technician of unquestionable ability had 69 birds which reacted to the tube method but to the whole-blood test only 45 of these reacted and the remaining 24 birds, which failed to react to the whole-blood test, were autopsied and 19 showed definite gross lesions of pullorum disease in the ovaries and pericardium and five showed no gross lesions.

The Whole-Blood Test

The tube method of routine testing in the hands of the laboratory technician and veterinarian has played a very important part in the control of baby-chick losses and the eradication of the disease from breeding flocks. However, pullorum disease continued to be the cause of enormous death-losses to the poultry industry in Indiana and it is the direct cause of the disturbance in 50 per cent of all chickens autopsied at Purdue University each year. Is the highly tooted whole-blood test proving to be as efficient as the tube test in identifying the "carrier" hen and, if so, why are these death-losses not being reduced? It is the genuine and highly efficient products that are being imitated and substituted. Can we progress on a sound eradication program in pullorum disease, a program that will protect and assure the baby chick buying public of getting what it expects, when the Federal Plan permits the use of such inefficient testing

methods and official grading which misleads the unsuspecting public?

The poultry industry is not making use of all the knowledge at hand in its efforts to control this highly infectious and transmissible disease, the spread of which can be prevented with good sound practical means, ultimately leading to eradication. If we, as veterinarians, would recognize the true nature of the disease in baby chicks, we would not prescribe medication for an affected flock, knowing that it is a self-limiting disease which runs its course, with the death-loss usually over in about three weeks. My prescribing usually includes the improving of any sanitary or hygienic conditions that may be possible, such as plenty of fresh air; clean, fresh and uncontaminated drinking water, and good feed at all times. We must not lose sight of our professional integrity and the veterinarian who prescribes and recommends medication for a pullorum disease-affected flock is just "sticking his neck out."

Coccidiosis

Next in importance to pullorum disease in young chicks is coccidiosis. This is a filth-born disease, caused by a protozoan, and is so general and widely distributed that not many poultry farms appear to escape an attack of coccidiosis each year. It is most frequently encountered between the ages of six and twelve weeks. Many forms of this disease are recognized in poultry today. Too much emphasis cannot be placed upon sanitation, when the brooding program is mapped out for the layers next winter. The "Six C" program, "Clean Chicks, Clean Ground, Clean Brooder-Houses, Clean Litter, Clean Feed and Clean Management," if followed religiously, will be found to have had a very important part in the general health condition when the pullets are ready to house.

I recommend that the brooder-house receive a thorough cleaning and disinfection. Clean, scrape and sweep out all visible evidence of former brooding. Scrub the floors and side walls with boiling hot lye solution, after which a thorough disinfection, with a government-approved disinfectant, is given the house. Then move this house into a field of alfalfa, blue grass or clover, preferably located so that it will not be necessary to walk through the yard where the old birds are kept to get to the little chicks, thereby eliminating the possibility of carrying infection from the old birds. A field where no chicks or chicken manure has been for at least two years is desirable. A good litter, four inches or so deep, of peat moss, ground corn-cobs or shavings

will serve very satisfactorily. Feeders and water founts should be placed upon frames made of 1 x 6-inch boards, placed upon edge, and covered with ¾- or 1-inch hardware cloth, so that the possibility of picking up or drinking contaminated material will be greatly lessened. The water founts placed upon these covered racks are especially beneficial in that the possibility of picking up coccidia is greatly lessened, because warmth and moisture are very essential to their growth and development.

Some years back, quite a large number of hatcherymen were the victims of high-pressure salesmanship when they thought they would lessen the possibility of their baby chicks developing coccidiosis if they dipped their hatching eggs in a solution before putting them in the incubators. This preparation proved to be entirely worthless in its intended purpose, for other research men proved that the coccidia would not survive 21 days in an incubator, no matter how high the humidity was carried. The only benefit the hatcherymen derived from that very disagreeable handling of hatching eggs was in the little "sales talk" value it had.

It is not difficult to make a diagnosis of coccidiosis in chickens. A few years back, we depended upon the evidence of bloody droppings. Such is not the case today. There seems to be many types of the disease recognized and bloody droppings are found only in the cecal type. When a diagnosis of coccidiosis is made in a flock of chicks, we should recognize and face the facts with which we are going to have to live. We know that no drug or combination of drugs will have any influence in effecting a cure. Why do so many of us persist in loading up our clients with worthless, bad tasting medication for these sick birds when sooner or later they find out that we were prescribing for their bank rolls instead of their sick chickens. Of course, many of the laity have great confidence in drugs and think they will cure any malady.

Food and Drug Administration Active

Every year there are many new discoveries announced for the cure of coccidiosis, when, as a matter of fact, Dr. H. E. Moskey, veterinarian for the federal Food and Drug Administration, in the enforcement of the pure food and drug laws, has removed from the markets all so-called remedies for this disease. Let us give careful consideration and thought to the condition with which we are dealing. A higher percentage of the flock seems to be affected, feathers are ruffled, there is great depression,

and autopsy shows extensive tissue destruction in the intestinal tract. The poultryman has brought us his problems, he is greatly worried as to the outcome in his flock of birds, he is much in need of assistance and is willing to pay us for our services. The knowledge that is ours of the value of medication for this condition surely will not prompt us to advise foul-smelling and unpalatable drugs in the drinking water or feed. Unquestionably such a course would be contraindicated.

Quite a few years back, I was privileged to hear an authority from the western coast lecture on this condition. That lecture has always been a guiding hand with me in coccidiosis problems. He had done some thorough and extensive research work in this disease, artificially infecting many groups of chickens with coccidia—large charge in the crop. The period of incubation was six days. He checked dried buttermilk with most all known cures at that time and found it gave by far the best results. Some of his flocks, experimentally treated with the "supposed to be specifics" for this condition, showed by far greater death-loss than the check flocks that received no treatment at all.

Dr. J. R. Beach, of the University of California, has reported the following comparison of dried buttermilk with other agents as a treatment for coccidiosis:

Buttermilk	14.5
Sweet skim milk	21.7
Control	38.7
Buttermilk	4.2
Condensed whey	17.1
Control	33.6
Buttermilk	11.3
Hydrochloric acid	32.1
Control	34.7
Buttermilk	13.5
Coccidiosis powder	43.2
Control	37.8
Buttermilk	12.6
Potassium dichromate	36.6
Control	33.8
Buttermilk	15.7
Ipecac	64.2
Control	40.3
Buttermilk	4.8
Bismuth subnitrate	27.7
Control	24.8

Experience has taught us that sanitation is the only effective means of controlling disease. So why is it not better to feed them a ration that is palatable, nutritious and easily digested

while they are sick? For a bird to have coccidiosis it must be constantly reinfecting itself, so if our sanitary measures will make it impossible for the bird to be picking up the organism, we will have accomplished something effective. Have the poultryman clean out the brooder-house where it is located, bed it with straw, then move it to clean ground and clean it out every other day. Confine the birds and keep everything as clean and dry as possible for these sick chicks.

Explain the life cycle of the organism to the poultryman, so he will better understand what his sanitary measures will accomplish. Feed the chicks a buttermilk ration for 8 to 10 days, consisting of 40 per cent buttermilk and 30 per cent each of corn and wheat. Allow no other feed during this period, but plenty of fresh, clean drinking water at all times. Stress sanitation. Lack of satisfactory results, if sanitary measures are carried out, can usually be traced to one of two things—either to a too low percentage of buttermilk or not feeding it for a sufficient period of time. In about four or five days, usually, the poultryman is greatly encouraged with the condition of his flock of birds. They are more active and have more "pep." Results are usually very satisfactory.

Charge for Autopsy Service

You veterinarians, no doubt, will wonder how I am able to buy "bacon and" with this line of treatment to my clients. I autopsy birds every day and charge $1.00 for that service. If, in my opinion, no medication is advisable and improved sanitation measures will accomplish more, that is my prescription. We all know that many of the diseases on the farm can be controlled better by sanitation, but, if once established, treatment seems to be of little benefit. We must all be sanitarians. It is a mistaken idea that we will lose all of our business if we preach disease prevention and sanitation to our clients, for on the contrary, they will know that we not only know our business but have their interest at heart. "True service will accelerate business."

We are able to control diseases in younger chicks in a fairly satisfactory manner. However, in older birds, with our present-day knowledge, disease control problems have not made very satisfactory progress. It is easy to recognize worms in mature birds and many of the diseases have been attributed to them as a cause, when many times other causes are responsible for the disease condition and death-loss.

Pullorum disease in turkeys has been recognized for some time. In the last few years turkey men have awakened to this

fact and are testing their breeding flocks with satisfactory results, and the test, when done by the serum agglutination method, compares with equal merit to the test in chickens.

Chicken-pox has made its appearance in our community, affecting turkeys as well as chickens. This is one disease that can well be prevented by vaccination of the young birds between the ages of one and three months, while they are still on the range, at least one month before they are put in the laying-house. If there has been no pox on the farm and none in the community, vaccination is not recommended. It is advisable only if there is a possibility of an outbreak. Pox is not a blood-stream infection. It is a skin disease, spread by contact or carriers. Never vaccinate a flock of pullets after they are in the laying-house unless there is grave danger of an outbreak.

Pullet year mortality and paralysis are two subjects not mentioned in this paper for there seems to be too much disagreement among investigators as to their causes for a common field practitioner to make a statement of any value bearing on these two sources of great economic poultry losses. No doubt in the near future, after more extensive research and careful investigation, these two baffling problems to the poultry industry will be answered.

The medical profession, in general, has been obliged to recognize the need of more accurate information on poultry diseases.

Eastern Iowa Veterinarians Go on the Air

The Eastern Iowa Veterinary Association went on the air during April, over Station WMT, Waterloo and Cedar Rapids, in a spring campaign to educate the farmers of eastern Iowa and western Illinois to a better and wider use of modern veterinary service.

The radio campaign, sponsored and paid for by the Association, consists of daily spot announcements during the noon hour, and will continue for three months. Timely subjects such as hog cholera, erysipelas, encephalomyelitis, rabies, scours, enteritis, and other live stock diseases are taken up in the broadcasts, and farmers are urged to consult their veterinarian promptly when any of the symptoms of such diseases occur.

Diamond Jubilee Convention
Hotel Pennsylvania—New York
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ENDOCRINES AND THEIR ROLE IN REPRODUCTION*

By W. G. VENZKE, *Ames, Iowa*
Department of Veterinary Anatomy
Iowa State College

"Endocrines and Their Rôle in Reproduction" is a title which would survey a vast field if all of the developments during the past few years were considered. Although endocrinology is a new branch of science, it holds its own, for the large number of writings upon its many phases, and for the complexity, confusion and disagreements existing in its vast literature. This paper is presented to point out some of the findings from research in this field and the interpretation which has been given them by leading investigators. This will include a brief discussion of the site of production, physiological function, and interrelationships of hormones concerned in reproduction.

The Pituitary

The observation of the effects of the removal of a gland of internal secretion is generally the most important step in the elucidation of the function of the gland. It was through this technic that the relationship of the pituitary body to reproduction was determined.

About twenty-five years ago, Cushing and his co-workers[1] and Aschner[2] demonstrated that removal of the pituitary body produced atrophy of the reproductive system. Smith[3] found that the principal changes in the genital tract of the hypophysectomized female rat consisted of an atresia of all follicles in the ovary and an abnormal persistence of corpora lutea. Estrous cycles and probably ovulation were said to fail. The uterus and vagina showed marked atrophy.

Parturition can occur in hypophysectomized rats, provided the gland is removed about two weeks before the termination of gestation. Hypophysectomy prevents implantation if performed not more than four days after copulation. Hypophysectomy performed between the seventh and 20th days of pregnancy results either in fetal death, resorption of feti or prolongation of gestation. Dead or living feti may then be born. Rats hypophysectomized on the 21st day of pregnancy have normal parturitions.

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

Why this prolonged gestation period should occur in some cases of hypophysectomy is not clear. The corpora lutea of pregnancy appear to be normal histologically, but the uterus is said to be less sensitive to the oxytocic principle of the posterior lobe.

In 1927, it was conclusively demonstrated that the anterior pituitary lobe maintained the activity of the gonads and through them influenced other reproductive organs. This conclusion was drawn from the work of Smith[4] and others who restored gonadal function after hypophysectomy by means of hypophyseal implants and induced precocious sexual maturity in normal females with implant treatment.

Hypophysectomy in normal male rats results in regressive changes of the testes involving the germinal epithelium and interstitial cells, so that a marked atrophy of the seminal vesicles and prostate occurs. Spermatozoa survive, judging by motility, about three weeks.

Pituitary implants were made into male hypophysectomized rats by Smith.[4] The atrophic genital tract was restored to normal size and appearance and normal secretory activity on the part of the interstitial tissue resulted. There was also a restoration of spermatogenesis, so that fertile matings occurred between normal female rats and the hypophysectomized male which received the homo-implants.

Gonadotropic Hormones

At present, gonadotropic extracts prepared from the anterior hypophysis do not possess the desired physiological purity. Results obtained from their use in the laboratory indicate that possibly two types of preparations can be made. One of these, if injected into a female, produces follicular stimulation; the second fraction, which has not been satisfactorily purified, produces luteinization.

1. *Follicular stimulator:* Much work has been reported on the fractionation of gonad-stimulating extracts of the pituitary by Fevold and his collaborators[5] and more recently by Wallen-Lawrence[6] and Evans and his co-workers.[7] A fraction has been obtained which produces pure follicular growth when injected into hypophysectomized female rats.

The work of Evans and Simpson[7] shows that a follicular fraction from the hypophysis injected into hypophysectomized male rats will produce a response in the seminiferous epithelium of the atrophied testes. Spermatids and spermatozoa were produced with slight or no response of the interstitial tissue and accessory reproductive organs.

2. *Luteinizing hormone:* At present the work upon the luteinizing gonadotropic hormone does not appear to be as conclusive as that of the follicle-stimulating hormone from the hypophysis.

Fevold and his associates[5] have secured a fraction from the anterior lobe which, if injected alone, is practically inactive for the stimulation of follicular growth, but is active for luteinization. It has very little action, if any, on the ovaries of immature rats but in rabbits medium-sized follicles already present in the ovaries are changed to corpora lutea. If injected together with the follicular stimulating preparation, it changes the developing follicles to corpora lutea in rats and rabbits. If this fraction is injected into normal or hypophysectomized male rats, an extensive interstitial tissue hypertrophy results.

Results of gonadectomy in the rat, guinea pig, rabbit and man appear to indicate that there is increased secretion and possibly storage of gonadotropic hormones by the anterior lobe.

It has been suggested that the production of ovulation in immature rats may depend upon a proper balance of follicular-stimulating and luteinizing activities and if an excess of the latter is effective, rapid luteinization will entrap the ova within the graafian follicle.

Other work by Hertz and Hisaw[8] has shown that ovulation occurred in 45 per cent of the young rabbits which were injected subcutaneously, first with follicular-stimulating and then luteinizing extract, over a period of three days for each extract. Ovulation failed to occur in young rabbits treated with unfractionated pituitary extracts, purified follicular-stimulating extracts, or luteinizing extracts alone.

Ovulation in immature rabbits appears to be efficiently produced by first growing the follicles by subcutaneously injecting follicular-stimulating extract. Following this treatment, suddenly make available a large quantity of the luteinizing substance by intravenous injection. In both rats and rabbits it was found that the injections of large amounts of luteinizing substance during the course of follicular development resulted in the formation of corpora lutea atretica without ovulation.

Thyrotropic Principle and Thyroid-Gonad Interrelationship

The pituitary and thyroid interrelationship was first suggested by Rogowitsch,[9] who observed enlargement of the pituitary of dogs and rabbits following thyroidectomy. Data so far gathered indicate that thyroidectomy has no important effect on total amounts of either thyrotropic or gonadotropic hormones in the

anterior lobe. The classic work by P. E. Smith[4] on hypophysectomy of the rat quite conclusively established that thyroid activity is dependent upon the stimulating action of the pituitary.

Following hypophysectomy, the thyroid becomes inactive and atrophic. This is indicated histologically by the flattened appearance of the epithelium and persistence of deeply stained colloid.

Smith[4] demonstrated that the atrophic thyroid of hypophysectomized rats could be restored to normal by the administration of fresh homoplastic implants of pituitary gland.

The interrelationship between the thyroid and gonads has been recognized for a considerable length of time as indicated by an increased frequency of goiter among humans during the period of puberty, pregnancy and menopause. The exact nature of this relationship is still questionable. Work of Abelin[10] and others shows that feeding of thyroid has an inhibitory effect on estrus.

Van Horn[11] presents work suggesting the possibility that the thyroid exerts an influence on follicular activity, and establishes a quantitative relationship between the thyroid secretion and that of the ovarian follicle. The real cause of the ovarian effect is not fully understood; it seems probable that the increased metabolism incident to a hyperthyroid condition is instrumental in eliminating a large amount of the theelin, thus keeping the amount in the body below the threshold value required for the production of estrus.

Weichert[12] observed that feeding 0.25 to 0.5 gram of desiccated thyroid daily to pregnant rats produced resorption of the feti in some animals, in other cases abortion or a prolonged gestation period with ultimate delivery of dead young. Thyroid administered to non-pregnant rats in comparable doses produced a diestrous condition after two estrous cycles, which was maintained as long as treatment was continued. He suggests this diestrus was due to the persistence of the corpus luteum brought about in some manner by the condition of the anterior hypophysis during hyper-thyroidism. It was also suggested that the delayed parturition was probably due to a deficiency of the estrus-producing hormone as a result of increased metabolism.

Da Costa and Carlson[13] find large doses of desiccated thyroid have a retarding effect upon sexual maturation in white rats, while small doses tend to produce an accelerating effect. Estrogenic substances injected into rats and guinea pigs for a period of a week to ten days enlarge the thyroid. If these injections

are continued for 20 days or more, an atrophic or involutionary effect is observed.

Parathyrotropic Principle and Parathyroid

The parathyroid glands are concerned with calcium metabolism. Only a few investigators have studied the effect of hypophysectomy on the parathyroid glands. The results of these investigations are conflicting.

Anselmino, Hoffman and Herold[14] have produced an enlargement of the rat parathyroid by administration of a pituitary extract. Hertz and Kranes[15] secured a fraction, by acid extraction of beef pituitary, which, upon injection into rabbits, produces mitosis and vacuolization of the parathyroid cells.

Anterior pituitary extracts have been said to produce an increase, or a diminution, or no change in the concentration of calcium in the blood.

The parathyroid gland is not of direct concern in reproduction, but it is known that the fetus consumes a large amount of calcium and phosphorus from the mother during gestation and lactation. Perhaps the parathyroid and accessory parathyroid glands assist in meeting these demands.

Adrenotropic (or Interrenotropic) Principle* and Adrenal Gland

P. E. Smith[4] has shown that hypophysectomy in rats results in a marked atrophy of the adrenal cortex. Homoplastic pituitary implants restored the cortex to normal size and appearance.

No very marked effect on the medullary portion of the adrenal gland has been described, nor has definite evidence for atrophy of the medullary portion been presented following hypophysectomy. Houssay *et al*[16] find in dogs a depletion of its epinephrine content following pituitary extract administration.

Britton and Kline[17] find adrenalectomized female rats seldom become pregnant. Pregnant animals which have been adrenalectomized fail to go through the normal process of parturition, abortion is frequent, and lactation fails to follow. Adrenalectomized male rats show reduced fertility compared to normal controls.

Brownell and his colleagues[18] have reported evidence for the presence of a second hormone in the adrenal cortex, "cortilactin," which is essential for lactation in the adrenalectomized animal.

*Evans points out in Chapter I of Glandular Therapy (Jour. Amer. Med. Asso., Chicago) that the principle commonly known in the literature as the "adrenotropic" is more accurately designated "interrenotropic" hormone.

Other work has shown maintenance of the salt balance in the adrenalectomized animal to be the only essential necessary for lactation.

Lactogenic Principle

In 1928, Stricker and Grueter[19] reported the production of lactation in pseudopregnant rabbits, before or after spaying, by injections of an extract of the anterior pituitary.

Later Riddle, Bates and Dykshorn[20] isolated the lactogenic principle, proposed the name, "prolactin," and described various methods for its isolation and concentration.

Lactation cannot continue in the hypophysectomized animal. If hypophysectomy is performed upon the lactating mouse, rat, or ferret, the secretion of milk ceases in approximately 24 hours.

There appears to be no evidence that the development of the mammary tissue is assisted by the lactogenic principle. The lactogenic principle apparently acts on fully developed mammary tissue to initiate and excite milk secretion.

Carl Moore and his workers[21] have proposed the hypothesis, based on experimental evidence, that the lactogenic principle is suppressed during pregnancy by estrogenic substances produced in large amounts during this period. At this time the ovarian hormones produce growth of the mammary glands, and at the time of parturition the placenta is lost, thus causing estrogenic substances in the blood-stream to drop suddenly, permitting the release of the lactogenic principle from the pituitary gland. The mammae, having been prepared during the meantime, then respond to the lactogenic principle with secretion of milk.

Experiments in pregnant and non-pregnant mice and rats suggest that the uterus may inhibit the secretion of the lactogenic hormone. Selye[22] concluded the act of sucking influences the secretion of the lactogenic hormone.

Posterior Hypophysis

Extracts from the posterior hypophysis exert physiological actions on the renal, cardiovascular, and respiratory systems largely through their effect on smooth muscle. Two fractions, "pitressin" and "pitocin," from the posterior lobe have been quite highly purified. Pitressin exerts physiological action on the renal, cardiovascular, respiratory and intestinal systems. Pitocin produces the oxytocic action which is of importance in this presentation.

From the work of a number of investigators it is clear that the nature and degree of reaction of the uterine musculature to the oxytocic principle is dependent upon the following: (1) species of animal, (2) whether the uterus is gravid or non-gravid, (3) phase of the estrous cycle (4) stage of pregnancy. The work done thus far with the oxytocic principle indicates it has no effect upon the human uterus during the early stages of pregnancy, due probably to the inhibitory effect of the corpus-luteum secretion. During the latter part of gestation and parturition, the uterus is easily stimulated by this substance. It is at this time that obstetricians find this principle useful.

After parturition, during the stage of uterine involution, injections of pitocin produce little or no response. This fact suggests that the estrogenic substances are necessary to sensitize the uterine musculature before pitocin will act.

Thymus Gland

Rowntree and his workers[23] have obtained some interesting results after injecting white rats of the Wistar Institute strain. Daily and intraperitoneal injections were made through successive generations of parents with a thymus extract prepared by Hanson.[24] Their results show that the young of the third and succeeding generations grow and develop at a very rapid rate, physically, sexually and psychically. For example, the testes for the sixth generation in the control rats descended in 35 to 40 days, while in the treated rats the testes descended in three or four days. In the females the vaginas of the controls opened in 60 to 70 days, while in the treated females the vaginas opened in 16 days. They also found that the thymus extract increased the fertility of adult rats. Adequate confirmation of these results is lacking as yet, but work is in progress.

Ovary and Its Hormones

Gonadectomy in mammals leads to the development of a type of individual which is intermediate between the two sexes. Ovariectomy before puberty results in complete failure of the accessory glands to undergo their normal development. Postpubertal ovariectomy results in cessation of all cyclic activity in the uterus, vagina, and mammary glands, but the actual degeneration of the reproductive organs is slow.

It is next necessary to consider the link in the chain of evidence that the ovary is an organ of internal secretion. As far

as the effects of ovariectomy are concerned, the ovarian control of accessory glands might be a nervous one, and it is necessary to show that the nervous connections of the ovary may be entirely destroyed without undesirable effects upon the accessory glands.

The effect of transplantation of the ovary is that large follicles of the transplant tend to undergo atresia and its corpora lutea degenerate. If good vascular connections are established, the transplant in the female undergoes growth, and cyclic changes characteristic of the normal ovary occur. Pregnancy from an ovarian graft has been recorded by Morris[25] and Grigorieff.[26]

If ovarian tissue is transplanted into males, the follicles appear to become cystic or atretic. Male guinea pigs with such grafts will secrete milk.

Estrogen Hormones

The collective term "estrogen hormones" includes those substances which have the ability to produce estrus. There are three widely known pure chemical compounds having estrogenic activity. They are theelin, theelol and dihydrotheelin. Estrogenic hormones were originally obtained from ovarian follicles. It has since been found that the human placenta and the urine of the pregnant human female contain large amounts of these hormones. The urine of stallions, pregnant mares and pregnant cows are also a source of these substances.

Estrogenic substances have many physiological functions. Certain secondary sex characters in the female are dependent upon the presence of estrogenic substances, such as plumage changes in certain birds, sexual swellings in the chimpanzee and baboon, and in monkeys reddening and swelling of the "sexual skin."

Allen and Doisy[27] concluded estrogenic substances would produce all the normal extra-ovarian symptoms of estrus in the ovariectomized rat or mouse.

Estrin injected into the normal pre-pubertal rat or mouse produces rapid proliferation of the vaginal epithelium and opening of the vagina. The uterus shows the typical estrous distension and development of a lower columnar type of epithelium.

Asdell and Marshall[28] report induction of early proëstrus changes in the anestrus dog by estrin injections.

Smith[29] showed early-stage pregnancy could be interrupted in the rat by injection of estrus-producing hormones, while Parkes and Bellerby[30] found pregnancy in the mouse could be terminated any time by adequate doses.

In the normal mouse and rat the injection of estrin immediately after copulation leads to the return of estrous symptoms,

and failure of embryo implantation. In the rat and mouse the estrus-producing hormone is not concerned with the production of post-estrus changes, as experimental prolongation of its activity is antagonistic to changes of the luteal phase. In the cow, Hammond[31] showed that removal of the corpus luteum after ovulation led to a much earlier return of estrus, therefore, the action of the corpus luteum is clearly directly opposed to that of the estrus-producing substance. In the rabbit uterus, prolonged estrus leads to none of the changes characteristic of pseudo-pregnancy. The rate and amplitude of the uterine contractions are controlled in part by estrogenic substances.

Estrogenic substances appear to have no stimulating effect upon the ovaries. These substances, injected into rats over long periods of time, may have a retarding effect presumably through their inhibitory action of pituitary secretion.

Estrogenic substances are responsible for growth of the mammary glands. The guinea pig at present is the only species investigated in which theelin alone will produce complete growth of both ducts and alveoli. Progesterone, the corpus luteum hormone, following the action of theelin, has been found necessary to produce complete mammary growth in the other species of mammals. Some recent work by Turner and associates[32] suggests that the theelin effect upon mammary tissue is not a direct one as heretofore believed, but that it acts through the pituitary gland. Theelin and theelol have been found to produce growth of the primary ducts of the rudimentary mammae of males. Theelin also induces growth in the epithelium covering the nipple.

Evidence that the maturing graafian follicle is not the causative factor in the production of estrus is forthcoming from the fact that maturation of the follicle begins after the estrus-producing substance has become active. The graafian follicle is not necessarily the only source of estrin. A quantitative examination has shown that estrin is practically equally divided between the follicles and the stroma of the ovary.

The mating instincts appear to be partly dependent on the ovarian follicular hormones.

The work described above has made it evident that the anterior pituitary body produces a substance or substances which have a potent action on the ovary, and it is reasonable to suppose that the anterior pituitary plays some part in regulating the ovarian cycle. It has been found that estrogenic substances, and of these particularly theelin, has a depressant effect on the secretion of gonad-stimulating substances from the anterior hypophysis.

Progesterone (Corpus Luteum Hormone)

After the follicle in the ovary ruptures and the ovum begins its journey down the fallopian tube, a temporary endocrine gland, the "corpus luteum," develops in the cavity of the ruptured graafian follicle. The corpus luteum then begins to send into the blood-stream a substance called "progesterone" that has the property of producing extensive development of the uterine glands. This uterine growth response is known as "progestational proliferation" and was first demonstrated by means of extracts in the rabbit endometrium by Corner and Allen[33] together. Hisaw *et al*[34] later showed that the uterus must first react to estrogenic substances before the corpus luteum extract can produce progestational changes.

When the fertilized ovum reaches the uterus, the endometrium is in a state of increased secretory activity, by which it is assumed nutritive substances provide for the development of the embryo. The uterus has also become sensitized and responds to the presence of the embryo by forming the maternal part of the placenta and the decidua. It is now well established that decidual growths are produced after traumatizing the uterus only in the presence of functional corpora lutea.

The work of several investigators has conclusively shown that removal of the corpus luteum in the early part of the gestational period in many species produces failure of implantation of the embryo, or, if the embryo has already been implanted, it produces abortion. There are some species, for example, the horse, where the presence of a corpus luteum is not necessary throughout pregnancy. It has been shown that during the last third of pregnancy in the mare only vestiges of corpora lutea are still present in the ovary. The ovaries have been removed during late pregnancy in the mare without abortion resulting.

The exact extent to which follicular maturation is inhibited by functional corpora lutea appears to show specific variation. In the guinea pig, Loeb[35] described waves of follicular growth even during pregnancy. Hammond,[31] by expressing the corpus luteum from the ovary of the cow, was able to cause the next estrous period to appear in the ninth day instead of the 20th day. In the cow, Hess,[36] Williams[37] and Tandler[38] have found that the persistence of corpora lutea results in sterility, as ovulation is suppressed. Expulsion of abnormally long-lived corpora lutea usually brings about the return of estrus. Definite information is lacking as to how the corpus luteum brings about suppression of estrus.

Several workers have shown that a premenstrual condition can be produced in the endometrium of the *Macacus rhesus* castrate monkey by injections of corpus-luteum extracts. The uterus must first be sensitized with theelin to get this reaction. A premenstrual endometrium is one showing extensive coiling and dilatation of the glands, an increase in size and secretory activity of the cells of the surface and glandular epithelium and practically no mitotic activity.

The effect of corpus-luteum extracts on the uterine musculature has been studied by Reynolds.[39] He finds that rabbits injected with such extracts show a temporary cessation of spontaneous uterine contractions (as measured through a uterine fistula). Excised rabbit's uterus *in vitro* has been used by Knaus,[40] who finds corpus-luteum extracts to inhibit the response of uterine muscle to the posterior pituitary oxytocic principle.

During pseudo-pregnancy in the rabbit and ferret, considerable development of the mammary gland takes place. This proliferation consists in the lateral extension and swelling of the ducts and the development of clumps of alveoli at the ends of the milk-ducts. Marshall[41] has described the development of the mammary glands of the dog during pseudo-pregnancy. In this animal the constructive phase progresses so far that the breakdown process at the end of pseudo-pregnancy actually results in lactation. Ancel and Bouin[42,43] removed the corpora lutea after sterile copulation, and were able to show that mammary growth during pseudo-pregnancy is entirely dependent upon corpora lutea.

Woodman and Hammond[44] and Asdell[45] have shown there are two phases of mammary development during pregnancy in the cow and goat. A development of the alveoli begins about midway through pregnancy and is correlated with a change in the type of secretion found in the mammary gland. Before this stage the secretion is serous in nature, characteristic of diluted milk. At the midway stage, the secretion changes to a thick pigmented fluid containing up to 40 per cent solids, which is mostly globulin. Drummond-Robinson and Asdell[46] found that the removal of corpora lutea in the goat did not allow for milk secretion, unless the corpora were removed after the globulin stage had been reached.

Relaxin

Hisaw[47] has prepared another substance, distinct from progesterone, found in the corpus luteum extract which he calls "relaxin." This preparation, following the action of theelin, has

the property of relaxing the symphysis pubis in the guinea pig. The relaxation produced is similar to that which occurs in the guinea pig during pregnancy.

Androgenic or Male Sex Hormone(s)

The testicle exercises at least two biological functions in the organism, the primary of which is maturation of male germ cells and the secondary, the secretion of one or more substances known as the testis hormone. The testicular hormone exercises its influence on the accessory glands of reproduction of the male.

McGee[48] was the first to extract the testis hormone successfully. There are three naturally occurring chemical substances which possess androgenic activity, namely, androsterone, dehydroandrosterone and testosterone. The question of whether or not the active principle obtained from the testis and urine of the male are identical substances is still an open one.

Administration of such substances as fresh pituitary tissue, extracts of dried pituitary or extracts of pregnancy urine produce a sudden increase in size and functional activity of all accessory reproductive glands at any period of the year. Wells[49] determined that hypophyses taken from seasonal breeding males at the approach of, or during, reproductive activity contained high potency for stimulating the gonads when implanted into immature female rats, but hypophyses removed during the period of sexual quiescence failed to exhibit this power.

It is well established that anterior pituitary secretions stimulate the gonads to function and maintain this function. It has also been demonstrated that testis hormone influences the pituitary to secrete lesser amounts of gonad-stimulating (gonadotropic) hormone. From this arrangement it may be assumed in the case of constant breeding males that the pituitary secretes gonadotropic hormone continuously at a very low ebb, being held in check by the inhibiting action of the gonadal hormone of the testis.

In seasonal breeding males the pituitary apparently does not secrete continuously. Perhaps outside agencies such as light duration, temperature and environment play a part in its activity.

The fundamental biological function of sex hormone is to condition the accessory reproductive glands to handle the germ cells in a manner to insure their proper meeting, and to condition the animal to respond at the appropriate time in a manner to insure propagation of the species.

It should be pointed out that from existing evidence testis hormone cannot be considered in any sense a stimulating substance for hypofunctioning testes. Rather than acting as a stimulating agent on the gonad, injections of testis hormone in sufficient concentration are injurious to gonad tissue. Gonad hormones are not gonadal stimulants, nor is the product of any specific endocrine gland a stimulating agent for that gland itself.

Gonadotropic Substance Occurring in Blood and Urine, Particularly During Pregnancy

In 1927, Aschheim and Zondek[50] reported that the urine of pregnant women contains gonadotropic hormone. The term "prolan" has been given this gonadotropic hormone. In 1930, Cole and Hart[51] found that the blood of mares in certain stages of pregnancy to contain large amounts of gonadotropic hormone. From a practical standpoint, the discovery of gonadotropic hormone in the urine and blood has offered a reliable method for the early diagnosis of pregnancy.

Zondek[52] and many others consider prolan to be a mixture of two gonadotropic hormones, "prolan A" and "prolan B." "Prolan A" stimulates chiefly the growth of the graafian follicle. "Prolan B" produces "luteinization" of the granulosa and theca cells of developing mature follicles. No adequate explanation has been given the relationship of these hypothetical substances to ovulation. "Prolan A" has been secured only from the urine or blood of nonpregnant, castrate or menopause women.

Small doses of prolan tend to produce only follicular growth; larger doses tend to produce hemorrhage into the follicle and the formation of corpora lutea with or without preceding ovulation. This relationship between doses and effect might indicate the presence of only one hormone or it may mean a different and higher curve of response to "prolan A" than to "prolan B." Proof that prolan is a combination of two or more hormones awaits chemical separation.

The gonadotropic hormone occurring in the blood of pregnant mares produces effects different from those characteristic of prolan, but resembling those of the anterior pituitary. Catchpole and Lyons[53] favor the belief that the hormone is secreted by the chorionic epithelium in the pregnant mare.

This gonadotropic hormone, when, according to Cole and Hart[51] and Evans *et al*,[54] administered to immature female rats, produces follicular growth and maturation far exceeding that ob-

served after the injection of prolan. Small doses may cause ovulation; large doses bring about luteinization without ovulation. In immature male rats the hormone stimulates Leydig's cells, and indirectly produces hypertrophy of the accessory reproductive glands. Prolan from pregnancy urine injected into immature male rats stimulates interstitial tissue with secondary effects on the accessory reproductive glands. "Prolan A" injected into immature male rats produces a stimulation of the seminiferous tubules. The gonadotropic hormone from urine of pregnant women does not stimulate the growth of the follicle in hypophysectomized rats, while the gonadotropic hormone of pregnant mare serum will stimulate follicular growth.

Gonadotropic hormone has been found in cases of hydatidiform mole and in uterine chorionepithelioma and appears to be identical with prolan or a fraction "B" of prolan. A similar hormone is excreted by men with malignant tumors of the testis. The gonadotropic hormone found in the urine of women with malignant tumors is somewhat different from prolan but may resemble the hypothetical fraction "A."

Endocrine Activity of the Placenta

The placenta stores or elaborates three principles: theelin, emmenin (Collip), and the anterior-pituitary-like principle (Collip).

Theelin is present in large amounts. The placenta probably takes over the secretion of this hormone during pregnancy.

Emmenin is an ester of estriol, producing its physiological effect when administered orally or by injection. Oral administration of emmenin produces estrus in immature rats in from three to five days. The ovaries are not definitely affected. No interference with the normal processes of pregnancy or lactation occurs from continuous daily administration. There is no action on adult castrates unless extremely large doses are given. It will be seen that the action of emmenin differs from that of theelin by lack of effect on the adult castrate (both male and female), and ability to produce an effect on the immature animal by oral administration.

The anterior-pituitary-like (A-P-L) principle acts similarly to the gonad-controlling principle of the anterior pituitary gland. It may be identical to Zondek's prolan from pregnancy urine.

Therapeutic Application

Therapeutic application of hormone preparations to endocrine disturbances is at the present time not well understood. There is much we do not know about hormonal action, standardization, and administration of extracts to give the effects desired.

The majority of standardization methods employed are biological in nature. An effect of a given preparation is determined by some physiological function serving as an end point which is measured either qualitatively or quantitatively in a test animal. At best, biological assays may contain errors ranging from 50 to 1,000 per cent. The effect produced in test animals that serves as the measure of potency may not be the effect desired or the effect that occurs in the animal which is treated clinically with the preparation in question. Differences in species also play a large part in making difficult the interpretation of assay data. Especially is this true with gonadotropic and estrogenic preparations. It must be emphasized that there are many differences existing in the physiology of the reproductive tracts of the various species of animals. Most of the products are assayed on mice, rats or rabbits. Thus, it becomes very clear that the stated activity of a great many hormone preparations may be considered as nothing more than rough approximations. Then, too, one cannot overlook the question of deterioration between the time of assay and use and the differences existing in rates of absorption and excretion of the active principle. All of these complicating factors do exist and make endocrine therapy with commercial products even more uncertain.

Therapeutic application with hormone preparations to reproductive disturbances of an endocrine nature have given variable results. The cardinal factor producing this variability is found in the fact that the preparations injected are not purified nor are they of consistent strength and composition. Preparations for therapeutic application are usually obtained by chemical extraction of the endocrine gland. This chemical treatment may result in degradation products and makes one wonder whether or not a principle obtained by extraction actually exists as such in the gland from which it is derived. A few substances have so far been obtained in crystalline form. The majority of the products are more or less contaminated with other active or inactive substances. Effects attributed to an active principle may be the result of its action with a relatively inert contaminate.

If proper application of hormones is going to be made, it is necessary to know more about the altered reproductive physiology in the affected animal. It is also very essential to know more about the responses of each specific kind of endocrine disturbance to different methods of therapeutic application. Too often the clinician recognizes but one symptom of an endocrine disturbance and then makes therapeutic application. For instance, he may recognize that a cow is not coming in heat or that she is constantly in heat. Without even making a thorough examination of the case, he proceeds with treatment. For successful treatment he must first determine the true etiology of the disturbance. At the present time there are too few men who can recognize a normal reproductive tract by rectal palpation. It is truly a difficult task and requires many hours of practice to train the finger tips so that they will give the true picture as it exists in the reproductive tract. After obtaining this mental picture through the finger tips, one must be able to interpret what it means.

One examination of a reproductive tract will tell very little. It is like looking at a photograph of a ball in the air—one cannot determine whether it is going up or coming down. A series of systematic examinations at regular intervals is necessary. Following this technic, it is possible to establish what is occurring in the reproductive tract. What every well managed breeding herd needs to obtain highest breeding efficiency is regular systematic examinations by competent veterinarians. As long as reproductive disturbances are treated without making a series of thorough, regular, systematic examinations and after treatment, failing to follow up with similar examinations, one will remain just an experimenter, hoping against hope that the condition will be relieved.

One becomes an investigator by first making a series of thorough, regular, systematic examinations of the reproductive disturbance and attempting, as far as possible, to determine the true etiology of the case. Then, after making proper therapeutic application, follow up the case with similar examinations and determine what actually occurred in the reproductive tract. At the present time, therapeutic use of hormones is still in an experimental stage; successes from their use on animals affected with reproductive disturbances of an endocrine nature are generally a result of chance rather than a result of application of established facts. From a clinical point of view, greater control over the results derived from treatment will be obtained, as

more knowledge is gained by a systematic study of endocrine disturbances.

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Bureau Transfers

Dr. Carl T. Loy (St. Jos. '23), from Omaha, Neb., to Perry, Iowa, in charge of meat inspection.

Dr. James A. Sullivan (Chi. '18), from Chicago, Ill., to Sioux Falls, S. Dak., in charge of meat inspection.

Dr. George E. Mitchell (Cin. '15), from Moultrie, Ga., to Memphis, Tenn., in charge of meat inspection.

Dr. Howard B. Fishback (McK. '17), from Perry, Iowa, to Erie, Pa., in charge of meat inspection.

Dr. John J. Burgess (Corn. '29), from Chicago, Ill., to South Saint Paul, Minn., on meat inspection.

Dr. Albert M. Snelling (Corn. '31), from Chicago, Ill., to Moultrie, Ga., on meat inspection.

GENERALIZED TUBERCULOSIS OF SWINE DUE TO AVIAN TUBERCLE BACILLI*

By WILLIAM H. FELDMAN, *Rochester, Minn.*
Division of Experimental Medicine
The Mayo Foundation

In an abattoir where the incidence of localized tuberculosis in swine attributable to avian tubercle bacilli was unusually high, I was impressed by the gross appearance of the morbid changes in the carcasses of swine that had been condemned because of generalized tuberculosis. I undertook, therefore, an investigation to ascertain the type of organism responsible for the infection in instances of generalized tuberculosis.

That the avian type of the tubercle bacillus plays an important rôle in tuberculosis of swine in the United States was demonstrated strikingly by the work of Van Es and Martin,[1] published in 1925. By tests for pathogenicity, these authors found the avian tubercle bacillus to be the infective agent in 88.51 per cent of 209 cases of tuberculosis of the lymph-nodes of swine. In 5.21 per cent of the cases, mammalian tubercle bacilli were present and a mixed infection with avian and mammalian tubercle bacilli accounted for the disease in 6.22 per cent. Van Es and Martin's findings have been amply confirmed by many other workers, so that at present the bacillus of avian tuberculosis is generally accepted as the microörganism responsible for the majority of cases of localized tuberculosis of the lymph-nodes of swine in the United States.

It is commonly believed among meat inspectors and animal pathologists that tuberculosis of swine, caused by the avian tubercle bacillus, is essentially a nonprogressive localized disease of the lymph-nodes of the mesentery and of the anterior portion of the cervical region. Although instances of widely disseminated or generalized tuberculosis in swine caused by the avian tubercle bacillus have been reported occasionally,[2-5] cases of tuberculosis in which lesions occur in the spleen, liver and lungs in addition to the usual involvement of the lymph-nodes have usually been attributed to the bovine form of the microörganism.

SOURCE OF MATERIAL AND METHODS

The material was obtained at a slaughtering establishment situated in the southeastern part of Minnesota. Most of the slaughtered swine came from the surrounding territory, where

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tuberculosis among chickens is extremely prevalent. The number of carcasses of swine retained in this particular abattoir on account of localized tuberculosis of the lymph-nodes is unusually large. In a period of eleven months, 117,963 swine were slaughtered, among which 21,246 (approximately 18 per cent) were tuberculous. Of the tuberculous carcasses 89 were condemned on account of generalized tuberculosis, while the disease in 85 was sufficiently severe to require that the carcasses be sterilized.

The material for the investigation was obtained, with two exceptions, from carcasses in which the disease was considered to be generalized and the carcasses, as a consequence, had been condemned as unfit for human consumption. The two exceptions consisted of material from carcasses which had been passed for sterilization. No attempt was made to obtain material from carcasses in which the nature of the lesions was considered unusual. Instead, practically every carcass condemned during the various periods of investigation was utilized for the study.

In most instances lesions other than those of the lymph-nodes were obtained. It was recognized that tuberculosis of swine may be due either to mammalian or avian tubercle bacilli or both. Although the finding of avian tubercle bacilli in the lymph-nodes of a case of generalized tuberculosis of swine would be presumptive evidence that the lesions of the other organs were due also to this form of the microörganism, it is more convincing if microörganisms of this type can be demonstrated in lesions in such organs as the lungs, liver, spleen or kidneys. Material was secured from 30 carcasses and consisted of seven specimens from the liver; 22 from the spleen; eleven from the lungs; two from the kidneys; four from the mesenteric lymph-nodes; one from a bronchial lymph-node, and one from a mediastinal lymph-node. In a large number of cases lesions from two different organs were studied.

Emulsions were prepared from the respective lesions and each emulsion, with one exception, was used for injection of two guinea pigs. In addition, portions of seven of the emulsions were injected intravenously into two rabbits each. Portions of the respective emulsions were also treated with 5 per cent oxalic acid and, after centrifugation, each precipitate was used to inoculate glycerinated and nonglycerinated egg-yolk-agar medium, which then was incubated at 37.5° C. When acid-fast bacilli developed, the material from the cultures was used to inject guinea pigs and rabbits. In each instance two guinea pigs were inoculated subcutaneously and two rabbits intravenously.

RESULTS

The results of the bacteriologic aspects of the study as well as the results of the tests for pathogenicity are summarized in table I.

It will be noted that tubercle bacilli were obtained on culture from all but four of the 30 carcasses from which the material

TABLE I—*Occurrence of tubercle bacilli in 30 cases of extensive or generalized tuberculosis of swine.*

CASE	CULTURE		PATHOGENICITY*				TYPE OF TUBERCLE BACILLUS
			GUINEA PIGS		RABBITS		
	TISSUE	RESULTS*	A	B	A	B	
1	Mesenteric lymph-node	+	+	+	+	+	Bovine
2	Spleen	+	−	−	+	+	Avian
3	Spleen	+	−	−	+	+	Avian
4	Mesenteric lymph-node	+	−	−	+	+	Avian
5	Lung	+	−	−	+	+	Avian
6	Spleen	+	+	+	+	+	Bovine
7	Liver	+	−	−	+	+	Avian
8	Mesenteric lymph-node	+	−	−	F†	+	Avian
9	Spleen	+	−	−	+	+	Avian
10	Liver	+	−	−	+	F	Avian
11	Spleen	+	−	−	+	F	Avian
12	Liver	+	−	−	+	+	Avian
13	Spleen	+	−	−	+	+	Avian
14	Lung	+	−	−	+	+	Avian
15	Kidney	+	−	−	+	F	Avian
16	Spleen	−					Undermined‡
17	Lung	−					Undermined‡
18	Bronchial lymph-node	+	−	−	+	F	Avian
19	Spleen	+	−	−	+	+	Avian
20	Lung	+	−	−	+	+	Avian
21	Lung	+	−	−	+	+	Avian
22	Spleen	+	−	−	+	+	Avian
23	Spleen	+	−	−	+	+	Avian
24	Spleen	+	−	−	+	+	Avian
25	Lung	+	−	−	+	+	Avian
26	Spleen	+	−	−	F	+	Avian
27	Spleen and lung	−					Undetermined§
28	Spleen and lung	−					Undetermined§
29	Spleen	+	−	−	+	+	Avian
30	Lung	+	−	−	+	+	Avian

*The plus and minus symbols indicate that the results were positive or negative.

†"F" implies failure in that the animal died too soon to indicate negative or positive results.

‡Guinea pigs injected with portions of original emulsions from tissue did not have lesions when killed.

§Rabbits as well as guinea pigs were injected with portions of the original emulsions from the tissues and none of the animals had lesions when killed.

was secured. In 24 (92.3 per cent) of the cases of generalized infection in which tubercle bacilli were demonstrated, the infective organism was the avian tubercle bacillus, and in two it was the bovine tubercle bacillus. In no instance was there evidence of a mixed type of infection.

COMMENT

Why tubercle bacilli were not demonstrated in four of the carcasses on culture or by inoculation of animals is not apparent. The specimens used in two of these cases consisted of lesions from the spleen and lung; in one, the lesions were from the spleen only, and in the fourth case lesions from the lung only were studied. Although in two of these cases only guinea pigs were inoculated with the emulsions from the tissue, in the other two, rabbits also were injected. The morbid changes in each of these cases had the characteristic, gross appearance of tuberculous lesions and it seems strange that attempts to demonstrate the etiologic agent by methods that proved satisfactory in other cases failed in these. Is it possible that unidentified acid-fast bacilli exist which are responsible for a tuberculous disease of swine? The failure of the bacteria to grow on a culture medium which is suitable for the ordinary forms of tubercle bacilli and the failure of emulsions made from morbid tissue to induce lesions in rabbits or guinea pigs can hardly be attributed to the infective agents being nonviable or avirulent. The lesions in the swine were of a generalized and progressive nature, suggestive of marked virulence or exceptional susceptibility on the part of the respective swine.

Cornell and Griffith[6] mentioned eight instances in which no growth was obtained on culture of submaxillary lymph-nodes from 103 swine. The same authors also mentioned the observations of Eastwood and Griffith, who failed to secure positive cultures from seven cases of typical localized tuberculosis of swine. Van Es and Martin failed to demonstrate tubercle bacilli in 31 of 250 tuberculous lymph-nodes of swine.

The presence of avian tubercle bacilli unassociated with bovine strains in a high percentage of cases of generalized tuberculosis should provide additional impetus to the consideration of adequate measures for combating tuberculosis of poultry. In addition to the immediate monetary losses as a consequence of condemnation of the infected carcasses, it is not unlikely that swine affected with extensive generalized tuberculosis of the avian type are in some instances capable of disseminating the infective agent to

healthy animals. Certainly an animal with numerous tuberculous lesions in the lungs, liver and kidneys is a much greater menace to other live stock than is an animal in which the disease is limited to the lymph-nodes.

The results of this investigation, although it embraces only a limited number of swine, indicate that in districts where the localized lymph-node type of tuberculosis of swine is due predominantly to avian tubercle bacilli, a considerable percentage of the extensive or generalized manifestations of the disease is caused by the same microörganism.

Summary and Conclusions

By cultural methods and by the inoculation of guinea pigs and rabbits, tissues from 30 swine which had extensive tuberculosis were examined to determine the bacillary type of the infective agent. In most instances lesions from tissues other than lymph-nodes were examined. Avian tubercle bacilli were found to be the sole etiologic agent in 24 (92.3 per cent) of 26 cases in which tubercle bacilli were demonstrated. In two cases bovine tubercle bacilli were obtained and in four cases the results were negative for tubercle bacilli. In 28 of the cases the disease was sufficiently severe to justify the condemnation of the carcass. In two instances the carcasses were passed for sterilization.

The results seem to warrant conclusions as follows: 1. Although avian tuberculosis of swine is predominantly a localized disease of the lmyph-nodes, the lesions may become distributed widely and may involve such organs as the liver, spleen, lungs and kidneys. 2. In districts where the incidence of localized tuberculosis of the lymph-nodes of swine due to the avian tubercle bacillus is high, the same microörganism is probably responsible for a considerable proportion of the cases of generalized tuberculosis in swine.*

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THE DIAGNOSIS OF FILARIASIS IN THE DOG*

By I. LIVE *and* E. L. STUBBS, *Philadelphia, Pa.*
School of Veterinary Medicine, University of Pennsylvania

In previous studies[1] it was found that an examination for filariasis in the dog may be made by examining the blood-serum for the presence of micro-filariae, and it was determined to be more advantageous to examine a drop of serum than a drop of whole blood for the detection of the filaria embryos. The microfilariae appeared to be more numerous in the serum, and they were more clearly visible in it, whether alive or dead, than in whole blood.

In order to decrease the possibility of missing positive cases with light infestations, it is advisable to use methods by which the number of microfilariae in a given blood sample are concentrated. The usual concentration method consists in drawing 1 cc of blood into a test-tube; adding 5 cc of a 2 per cent acetic acid solution; mixing it thoroughly; centrifuging the mixture for three to five minutes; decanting the supernatant fluid; transferring a drop of the well-mixed sediment to a slide; and examining it under low-power magnification for the presence of dead microfilariae. According to Morris *et al*,[2] comparative examinations by means of the drop method and concentration method of whole blood on 100 dogs revealed 21 per cent positive cases when the concentration method was employed, and only 10 per cent positive cases with the use of the drop method. Thus the drop method with whole blood was subject to 50 per cent error, as compared with the concentration method of whole blood.

In this study an attempt was made to determine the efficiency of concentrating microfilariae in a given sample of blood-serum as compared with the concentration method using the same amount of whole blood. The following method was employed: About 5 cc of blood was drawn from a superficial vein and 0.5 cc of the uncoagulated blood was immediately discharged into a small test-tube which contained 5 cc of a 5 per cent glacial acetic acid solution. The remainder of the blood was poured into a vial where it was allowed to coagulate. After the serum had separated from the clot, 0.5 cc of it was measured into a small test-tube which contained 5 cc of a 5 per cent glacial acetic acid solution. Both sets of tubes, those with whole blood and those with serum, were inverted several times to mix their contents, and then were centrifuged at moderate speed for ten minutes.

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Subsequently, the supernatant fluid was poured off; except one or two drops, in which the sediment was resuspended. This mixture was then poured on a microscopic glass slide. The whole smear was examined systematically and the number of microfilariae was counted. While the microfilariae were clearly seen in smears from the serum samples, they were less distinctly perceptible in those from the whole blood samples. In the latter the erythrocytes were broken down and dissolved by the acetic acid, yet a certain amount of débris was left which, together with the microfilariae, settled to the bottom of the test-tube during centrifugalization and, when transferred to a slide, it obscured to a certain extent the view of the microfilariae. A comparison of the counts showed that there were many more microfilariae in the serum than in the same amount of whole blood from the same dogs.

TABLE I—*Comparative counts of microfilariae in 0.5 cc of whole blood, and in 0.5 cc of blood-serum taken from the same dogs.*

DOG	MICROFILARIAE IN WHOLE BLOOD	MICROFILARIAE IN BLOOD SERUM
1	75	98
2	11	75
3	10	17
4	8	9
5	0	12
6	70	207
7	47	77
8	6	24
9	7	21
10	64	170
11	1	1
12	1	10
13	21	60
14	4	15
15	1	4
16	5	13
17	28	110
18	11	72
19	4	5
20	16	59
21	14	45
22	32	41
23	75	73
24	1	15
25	10	14

With the exceptions of dogs 11 and 23, the counts from the serum were higher than the counts from whole blood. The total number of microfilariae counted in the serum samples from the

25 dogs was about two and a half times as high as the number counted in the samples of whole blood. In dog 5, a positive case would have been missed if only the whole blood concentration method had been employed.

Routine Concentration Method with Blood Serum

In routine examinations for filariasis in our laboratory, 3 to 5 cc of blood is obtained from a superficial vein with a sterile syringe and needle, placed into a vial and allowed to clot. After the serum has separated, in the course of about two to three hours, it is poured into a test-tube (there is about 1 cc of serum), 5 cc of a 2 to 5 per cent glacial acetic acid solution is added, the tube is inverted several times to mix its contents, and is centrifuged at moderate speed for ten minutes. Then the supernatant fluid is poured off carefully in one stream without disturbing the sediment at the bottom of the tube, and only about two drops of fluid are left in which to resuspend the sediment. After the test-tube has been shaken several times and the sediment is resuspended (the sediment is not visible grossly), the tube is inverted and the contents are poured on a glass slide. The microfilariae are dead, because of the action of the acetic acid, and they appear under the low-power magnification of the microscope as thin, fiber-like structures tapering at one end. Under high power, granulated material is seen in the central portion.

If a centrifuge is not available, the same technic may be employed, but instead of centrifuging the serum-acetic acid mixture, it is allowed to stand at room temperature overnight. Then the supernatant fluid is poured off, and the sediment is examined in the manner described above. Counts of microfilariae in centrifuged serum samples from 20 days and in the corresponding serum samples which had been allowed to stand at room temperature after the acetic acid had been added, showed that the number of microfilariae counted in the latter after standing, as short a time as four hours compared very well with the number found in the centrifuged samples. The acetic acid solution acts as a preservative, and the microfilariae will remain intact in it for a long time.

In case one does not possess a centrifuge, the second method of examining blood-serum for microfilariae is to be highly recommended in preference to the examination of one or several drops of blood, or serum, on a slide. In many instances, when the microfilariae were not very numerous in the blood, they were not

found with the drop method but they were detected with the concentration method.

Measurements of Microfilariae and of Larvae of Ancylostoma Caninum

Frequently the remark is heard that in making blood examinations for microfilariae one might mistake hookworm larvae, circulating in the blood-stream, for microfilariae. Aside from the fact that the structure and shape of the two is markedly different, there is a considerable difference in size between them. A number of microfilariae from dogs' blood were measured, and they averaged 280 microns x 5 microns. Ancylostoma eggs from dogs were obtained and allowed to hatch into full-grown larvae. The measurements of the larvae were 610 microns x 26 microns. Measurements given in standard text-books for microfilariae are 285 microns x 5 microns and, according to Neveu-Lemaire,[3] the larvae of *Ancylostoma caninum* measure 560 microns x 28 microns.

The above figures show clearly the difference in size between the two, and no one acquainted with the appearance of microfilariae should ever confuse them with hookworm larvae. Furthermore, in our own experience in the examination of many hundreds of blood samples from dogs, we never found Ancylostoma larvae in the blood, although many of the dogs were known to be heavily infested with hookworms, and had opportunity to ingest many infective larvae.

Summary

1. The concentration method is preferable to the drop method when blood-serum is examined for the presence of microfilariae.

2. The use of blood-serum is preferable to the use of whole blood, when the concentration method is employed, because the microfilariae are more numerous in the serum than in the same amount of whole blood, and they are more clearly seen in the former.

3. If a centrifuge is not available, the number of microfilariae in a sample of blood-serum can be concentrated by mixing it with acetic acid solution, and allowing it to stand overnight.

4. A 2 to 5 per cent acetic acid solution will preserve microfilariae for a long time.

5. Measurements of microfilariae of *Dirofilaria immitis* show that they are only half the size of *Ancylostoma caninum* larvae, and there is little chance of confusing one with the other.

6. No Ancylostoma larvae were found in the blood of several hundred dogs examined for filariasis.

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State Board Examinations

Kansas State Board of Veterinary Examiners. Kansas State College, Manhattan, Kan. May 26, 1938, at 8:00 a.m. Dr. Thos. P. Crispell, Secretary, Exchange Bldg., Parsons, Kan.

Iowa Veterinary Medical Examining Board. State Capitol, Des Moines, Iowa. June 14-15, 1938. Applications must be on file at the Division of Animal Industry not later than 8:00 a. m., on June 14. Further information may be obtained from Dr. H. A. Seidell, Secretary, State Capitol, Des Moines, Iowa.

California Board of Examiners in Veterinary Medicine. Los Angeles, Calif. June 20-22, 1938. Applications must be on file with the Secretary at least 30 days prior to date of examination. Dr. Nelson E. Clemens, Secretary, 183 Castro St., Hayward, Calif.

Nebraska Bureau of Examining Boards. State House, Lincoln, Neb. June 21-22, 1938. Applications must be on file at the Bureau at least 15 days prior to date of examination. Mrs. Clark Perkins, Director, Bureau of Examining Boards, State House, Lincoln, Neb.

North Carolina State Board of Veterinary Medical Examiners. Robert E. Lee Hotel, Winston-Salem, N. C. June 27, 1938. Dr. P. C. McLain, Secretary, Route 4, Charlotte, N. C.

Florida Board of Veterinary Examiners. Jacksonville, Fla. June 27-28, 1938. Dr. H. C. Nichols, Secretary, Box 405, Ocala, Fla.

Oklahoma Board of Veterinary Medical Examiners. State Capitol Building, Oklahoma City, Okla. June 29-30, 1938. Applications may be obtained from Dr. Samuel R. Espy, Secretary, 125-127 West 23rd St., Oklahoma City, Okla.

Diamond Jubilee Convention
Hotel Pennsylvania—New York
July 5-6-7-8-9, 1938

TORSION OF THE COLON*

By CHARLES HAASJES, *Shelby, Mich.*

The owner of a ten-year-old black Percheron gelding found the horse sick at about 8 o'clock in the morning. The animal was exhibiting signs of slight abdominal pain and was getting up and down frequently. The owner turned the horse into the barnyard to give him more freedom in his actions.

When I arrived upon the scene at about 9 o'clock, the horse was rolling in the barnyard and was somewhat uneasy. Auscultation and palpation of the abdomen were negative. The temperature, pulse and respirations were normal. I made an exploratory examination of the abdominal cavity *per rectum* for the purpose of determining whether there was a torsion of the colon. Nothing abnormal was found, not even an area of tenderness. My diagnosis was "colic."

The horse was treated and was somewhat easier about 30 minutes later. I had to leave to attend another case but made a second call the same day at about 3 o'clock. The horse was worse in every respect. There was no peristalsis, the pulse was 94, the respirations 37, and the temperature 104.4° F. On account of the violent movements of the animal, I was unable to make as thorough an examination as I would have liked to.

A slight tympanites was relieved with the trocar but this did not relieve the pain. Bloating immediately returned. I gave 10 cc of cannabis indica intravenously and after the patient quieted down, I made another rectal examination. My hand immediately came in contact with a cord-like, painful mass, just anterior to the brim of the pubis. I made a diagnosis of torsion of the colon and gave an unfavorable prognosis.

In my opinion, the torsion had occurred recently. Therefore, I tried rolling the patient. I committed a serious error by hog-tying the horse while he was down, thinking that we could roll him over on his back, but unfortunately his legs extended too

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far from his body for him to be rolled over on the breast. I should have known enough to use casting harness, so that I could have tied him, as for the ridgling operation. Then we could have rolled him continuously in one direction. The patient died and the postmortem revealed a torsion of the colon.

My esteemed colleague, Dr. J. W. G. Hanson, of Greenville, Mich., at a meeting of the Western Michigan Veterinary Medical Association, several years ago, in a very scholarly paper on "Displacement of the Intestines," conveyed the impression that torsion of the colon is invariably a primary condition. However, in the case under consideration, the evidence seemed to indicate that the torsion had been a complication of an attack of colic, while the animal was rolling in the barnyard.

The work of the veterinary practitioner is immensely interesting because he is dealing continuously with things along the border-line between the known and the unknown, and if he will persistently study the unknown, along with the proper observation, he need not have a dull minute in his whole life.

LUNG WORMS IN A KITTEN*

By H. D. PRITCHETT, *Philadelphia, Pa.*

Lung worms of the cat are reported by Cameron[1] as being a "fairly common parasite of cats in certain parts of Britain and Europe, and have been recorded from the U. S. A." Cameron also "showed that the lung worm of the cat was transmitted by mice, in which it underwent a development cycle," the mouse becoming infected by eating the feces of infected cats which contained the larva; the mouse in turn was eaten by the cat, which was infected thereby.

The writer was afforded an opportunity to autopsy a kitten (from Charlotte, N. C.) accompanied by such a detailed and interesting history that a record of the same was thought to be of value. So that a better appreciation of this case may be had, it would seem justifiable to include in this report, jointly, the histories of three feline individuals.

A cat, born and raised until about four months old on a small dairy farm (in Virginia) known to be infected with infectious abortion, after aborting her first kittens, delivered four living and normal progeny at the age of two years. Three of these

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kittens were disposed of to distant friends of the owner; the fourth one, being a male, was retained and becomes the subject of this report. The adult cat, while never being especially thrifty until at about the age of 1½ years, nevertheless required medical attention on only one occasion, previous to that described herein. This was for the treatment of round worms which were vomited; treatment was administered by the local practittioner.

Just prior to the death of the kitten, which occurred at the age of six months, the adult survived a period of severe undiagnosed illness. Throughout this period, the temperature fluctuated between 104.4 and 106.2° F. and continued to do so for approximately ten days. Anorexia, extreme prostration and emaciation, with anemic mucous membranes in conjunction with evidence of pain when the thorax was compressed, constituted the entire manifestation of symptoms. Auscultation revealed no evidence of thoracic disease. All three cats are known to have been passionately fond of slugs, lizards, rats and mice, grasshoppers, roaches, and other insects, in addition to their customary diet.

The above syndrome occurred almost simultaneously with that of the kitten in question. As the third feline died under identical conditions (within four days of the kitten's demise), there was thought to be some analogy between these three cases. However, the adult cat completely recovered with the routine treatment and other procedure which is not describable at this time.

When the kitten was found dead, it was reported to have appeared as though it died quietly, there being no evidence of a struggle. Nevertheless, the owner feared it had been poisoned, as she was certain that local animosity existed; and she requested a diagnosis of the writer.

The history submitted by the owner would indicate the existence of an acute elevation of temperature in life, as the animal was very languid for several days and radiation of heat from its body could be detected. The kitten's breath was noted to be very unpleasant on several occasions at irregular intervals. About eight weeks before its death, it suffered a brief illness, at which time the temperature was 105.0° F., which subsided with the usual purgative treatment.

When the cadaver was presented for autopsy, rigor mortis was complete. The mouth was open, and all visible mucous membranes were cyanotic, but there were no discharges from any of the natural body openings. Neither was there evidence of traumatism. In life, the animal had made rapid growth and was well developed physically.

The autopsy findings were rather striking, as no alterations of any description could be detected in the abdominal viscera. The only gross changes that occurred within the thoracic cavity were confined exclusively to the lungs, no other adjacent organ or tissue being involved. This fact incited much curiosity on the part of the autopsist.

The lungs were extremely hemorrhagic in appearance, with areas of cyanosis, and scattered throughout their visceral surfaces appeared many hard nodules, approximately the size of a No. 4 bird-shot. This organ was not incised. Therefore, the occurrence of these nodules throughout the parenchyma was not observed. While tuberculosis was hardly a logical conclusion in consideration of the physical condition which this animal possessed in life, and its age, it was given some thought—as the kitten was raised in a home with a child suspected of having (but not diagnosed) active pulmonary tuberculosis. There were no exudates within this cavity, neither were there any pleuritic adhesions. Fecal examinations were negative.

The lungs and heart were removed *en masse*, also the diaphragm, all of which were refrigerated for 24 hours. The specimens were packed in borax, and submitted to the Pathological Division, U. S. Bureau of Animal Industry, with a request that they be examined for "Bang's disease, tuberculosis (which type, if present), trichinosis, or any other diseases which this history might suggest; also a histological diagnosis." Excerpts from the report[2] are quoted herewith:

> The heart-blood of the kitten gave negative results to rapid plate test for Bang's disease.
>
> The Zoölogical Division reports the presence of lung worms, *Aleuro-strongylus abstrussus*. Section of this parasite could be seen in the lung tissue on histological examination. No parasites were found in the muscle tissue of heart or diaphragm.
>
> Histologically, the lung tissue showed extensive chronic pneumonic changes with almost complete solidification, possibly due primarily to the parasitic invasion. There was also considerable calcification in the region of the lung parasites. It was also noted that there was extreme thickening of the arterial walls, resulting in almost complete obliteration of the lumen of some of the vessels. In addition to the pneumonia, there was also observed an adenomatous condition of the lung tissue in some areas, the neoplastic cells apparently having their origin from the bronchial epithelium. No evidence of tuberculosis was found in the lung tissue.
>
> The death of the kitten was evidently the result of the advanced chronic pneumonia changes.

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AN OUTBREAK OF FOWL TYPHOID IN GUINEAS*

By F. R. BEAUDETTE, *New Brunswick, N. J.*
New Jersey Agricultural Experiment Station

On September 18, 1937, two dead guineas were received from a farm at Cedar Grove, Essex County, New Jersey. The birds were in good flesh, and autopsy showed dusky lungs, acute swelling and mottling of the liver, acute swelling of the spleen and a catarrhal enteritis. Both birds showed mucus in the proventriculus. A pure culture was isolated from the liver of each bird.

The farm was visited on September 20, and it was learned that 100 day-old guineas had been placed in a house with twelve ducks of the same age. There had been no losses until about two weeks before, when the guineas began dying. In the course of two weeks, 91 guineas had died and one duck may have died. According to the owner, the birds seemed to choke up, the head parts became bluish and death occurred very soon thereafter. The remaining nine guineas were quite inactive and occasionally one would shake its head and cough. These birds were now about three months old. Elsewhere on the farm, there were about 1,500 chickens, 145 turkeys and a dozen geese in which no unusual losses had occurred in spite of the fact that an occasional chicken got into the infected pen. There were also about 100 very young guineas elsewhere, which had had no direct contact with the infected lot and suffered no losses.

A dead and a sick guinea were brought back to the laboratory for examination. These showed about the same changes as the previous two. The spleens were much enlarged and friable. The livers were enlarged and friable. The lungs of one were congested and of the other dusky. There were no hemorrhages on the heart. One showed catarrhal enteritis with no reddening and in the other enteritis was less pronounced. One bird had a large amount of mucus in the nasal cleft and some in the upper trachea. There was little mucus in the cleft of the other and none in the trachea. Cultures from the liver and spleen of the first and from the liver, spleen and kidney of the second yielded a pure growth. A culture from the nasal cleft of the second bird which showed less mucus also gave several colonies of the organism in question.

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A final guinea was received on September 25, which showed the usual enteritis, acute swelling of liver and spleen and edema of the lungs. A pure culture was obtained from the liver.

At the time of isolation, the fermentation reactions of each culture were studied in dextrose, lactose, maltose and saccharose. All were alike in that they produced acid in dextrose and maltose but not in lactose or saccharose. Later, the liver cultures from the first two birds, the spleen culture from the third bird and the nasal culture from the fourth bird were inoculated into Dunham's fermentation tubes with Andrade's indicator. Acid was promptly produced in levulose, mannitol, galactose and dulcitol. Slight acid was produced in the inner tube after 48 hours in rhamnose by liver and spleen cultures, the nasal culture gave very slight acid after ten days of incubation and the other liver culture was negative. Trehalose was fermented promptly by one liver culture. The other liver culture and the nasal culture produced acid after 96 hours, while the spleen culture produced it only after ten days. In xylose slight acid was produced in 96 hours by one liver culture, while the other failed to produce acid. The spleen and nasal cultures produced acid only after ten days. Acid was produced in glycerin by the four cultures in 96 hours. The spleen culture produced a slight acid reaction in the inner tube in arabinose; the three other cultures were negative.

Curiously enough, dextrin, which is supposed to be fermented by the typhoid organism, was not attacked by any culture.

All four cultures were uniformly negative in soluble starch, sorbite, salicin, inulin, adonitol, melezitose, amygdalin, inosite, raffinose and erythritol.

Colonies rubbed up in positive pullorum serum showed agglutination.

A review of the literature fails to reveal a similar outbreak in this species. The outbreak is unique for this disease in any species, in that it provoked a known mortality of 92 per cent in less than three weeks. The failure of the young ducks to contract the disease even though in constant contact is rather remarkable but to be expected. Similarly, the failure of chickens to contract the disease is also remarkable because some of them did get into the infected pen from the adjoining pen.

It is well known that chickens are frequently attacked by typhoid. Outbreaks in turkeys are not uncommon, as reported by Hinshaw.[1] Pfeiler and Roepke[2] list the turkey, pheasant and guinea as susceptible but claim that ducks, geese and pigeons are not. Donatien *et al*[3] claim that the fowl, turkey, guinea and

peacock are susceptible, but that ducks and geese are immune. According to Kaupp and Dearstyne,[4] turkeys are less susceptible than chickens, and guineas are slightly susceptible but yield to artificial infection. They also claim that ducks and geese are resistant. These authors fed infection to a guinea with the result that it showed symptoms on the second day and died on the fifth. From an outbreak in parrots in the Philadelphia Zoölogical Gardens, Fox[5] claims to have isolated *"B. sanguinarium."*

As regards experimental infection, Kaupp and Dearstyne report that it is possible to kill the English sparrow by subcutaneous injection. The goose is not killed by subcutaneous or intramuscular injection, according to Pfeiler and Rehse.[6] These same authors were unable to infect ducks by feeding, intramuscular or subcutaneous injections, but Pfeiler and Roepke inoculated a duck which died six days later. A duck fed cultures by Kaupp and Dearstyne showed symptoms on the fifth day but recovered on the ninth. Notwithstanding these reports, te Hennepe[7] records the finding of Klein's disease eight times in 268 duck autopsies.

Pigeons have frequently been used as subjects for experiments. According to Klein,[8] subcutaneous injection is without effect and Lucet[9] was unable to infect pigeons with 1 cc subcutaneously. However, Moore[10] killed a pigeon in eight days by a subcutaneous dose of 2 cc of broth culture. Pfeiler and Roepke injected 1 cc of a 24-hour broth culture subcutaneously into a pigeon on April 22 and it died May 3, but its heart-blood was not infectious for a second pigeon. Two pigeons were given doses *per os* by Kaupp and Dearstyne, and although the pigeons sickened on the third and fourth days, respectively, they recovered by the 15th. By the intravenous route, Moore killed a pigeon in four days with a dose of 2 cc and in six days with 1 cc. Pfeiler and Roepke injected an agar suspension intramuscularly on April 4 and the pigeon died May 30. One cc of a 24-hour broth culture injected by Kraus[11] killed a pigeon in four days. Pfeiler and Rehse also report that a large dose intramuscularly caused death in one pigeon in seven days, but usually is without effect. According to te Hennepe and Van Straaten,[12] pigeons are not always killed by the organism (mode of infection not given) and Donatien *et al* claim that pigeons resist inoculation.

The above review does not include the results of inoculation of mammals.

An interesting point in connection with the outbreak in guineas is that affected birds showed respiratory symptoms. This is cor-

related, too, with the finding of mucus in the nasal cleft and trachea, and rather severe congestion of the lungs. Moreover, the organism was recovered from nasal exudate. Gauger[13] has reported a similar isolation in chickens and even established a carrier state in this species.

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AN UNUSUAL TREATMENT FOR STRYCHNINE POISONING*

By J. J. CATTANACH, *Newark, N. J.*

Owing to the present popularity of the dog, I consider it my duty to report the following, although many years have elapsed since I had this strange experience.

In the spring of 1898 (I was then practicing with my father and brothers in New York City), I received an emergency call one night from the Koster & Bials Theatre. On my arrival, I found a Maltese poodle lying dead, and eight other dogs of the same breed in the throes of strychnine poisoning. In those days, dog practice was at a minimum and I had nothing in my medicine-case suitable for such small animals. Realizing the rapidity with which strychnine acts, I was at a loss to know what to do,

*Received for publication, January 12, 1938.

but chance came to my rescue and, to this day, I do not know what made me do what I did.

Mechanically picking up one of the dogs by the neck, and at the same time grasping the loins, I began a bilateral bending movement of the spinal column, in and out, at the speed of a slow-ticking clock. This gave me the sensation of bending a soft lead pipe. In the short time of about ten seconds, I felt the body suddenly relax and become limp. I put the dog on the floor and he walked around in a semi-dazed manner, but soon became normal. I treated all the other dogs eight in number, by this same movement and they all made perfect recoveries. They were resting peacefully when I was about to leave, which was about 1½ hours after I first saw them.

It was dumb luck on my part and I do not claim anything for making this discovery, as it is beyond me and I cannot fathom it, but I do know that it is an actual fact and that strange things happen.

A peculiar thing about this case was that none of the dogs vomited and none had had any other treatment than the method I employed.

Many of the wonderful cures today can be traced to experiments on the lower animals, and it may be possible to treat certain nervous diseases in the human being by the same method I used, but it is up to those engaged in research work to decide.

Now comes the other side of my story, which may be of interest. Professor Dougherty, who was exhibiting these wonderful dogs, after the treatment said, "Your diagnosis was wrong. Who would have the heart to give those dogs strychnine? Why they are really human." I then asked him if he had given the dogs any medicine. "Yes," he answered, "I gave them pills before their act to quicken them in their work." I advised him to have the pills analyzed, which he did. He reported two days later that each pill contained 1/20 grain of strychnine. He bought the pills from a certain veterinarian, who claimed that he gave his formula to a certain company for the manufacture of the pills, each one to contain 1/120 of a grain of strychnine. As the result of this accident, the professor sued the veterinarian and the pill company, jointly, for $10,000.00 for the loss of one dog and the breaking of his theatrical contract. I was told he received a verdict for the full amount.

Diamond Jubilee Convention
Hotel Pennsylvania—New York
July 5-6-7-8-9, 1938

PSEUDOTUBERCULOSIS IN A DEER*

By W. J. BUTLER, *Helena, Mont.*
State Veterinary Surgeon of Montana

The accompanying photograph (fig. 1) shows the head of a deer affected with pseudotuberculosis. The deer was brought to the Montana Livestock Sanitary Board Laboratories on March 15, 1938, by State Game Warden James Weaver and a rancher by the name of Ed Morgan. The animal was killed in the Sal-

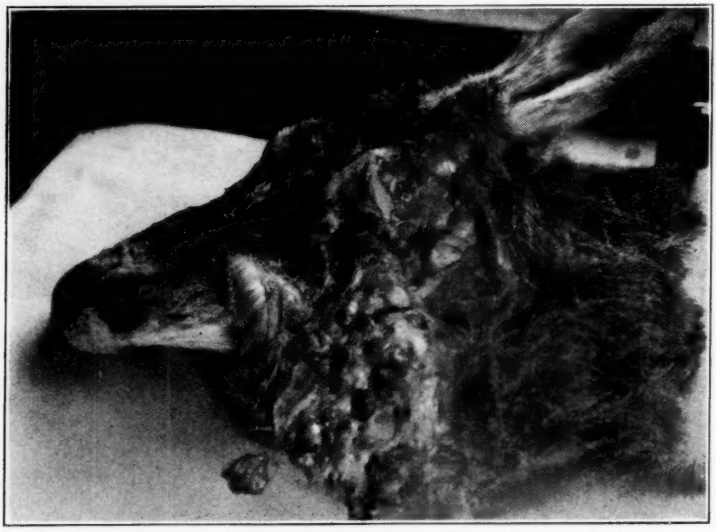

FIG. 1. Head of deer affected with pseudotuberculosis.

mon Lake country. Mr. Morgan observed that it was very weak and had a visible swelling on both jaws. The animal was shot, to put it out of its misery. There were numerous abscesses containing a thick, greenish-yellow pus found on both sides of the head and in the mediastinal glands. Laboratory examination proved the causative agent to be the *Corynebacterium ovis* (Preisz-Nocard bacillus).

*Received for publication March 21, 1938.

Diamond Jubilee Convention
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SOME FUNDAMENTAL INVESTIGATIONS ON THE RESISTANCE OF TUBERCLE BACILLI. H. J. Cooper and Maurice L. Cohn. Jour. Bacteriol., xxxv (1938), p. 223.

In studying the thermal and acetic acid resistance of human tubercle bacilli, it was found that the type of media on which the bacilli had been cultured was a determining factor in the resistance of those bacilli. Those cultured on egg media containing broth usually showed a greater resistance to heat and acetic acid than those cultured in the absence of broth. Bacilli cultured on whole-egg medium with broth showed a greater resistance than those cultured on glycerol-broth agar. The age of the cultures, within certain limits of from three to six weeks, appeared to exert no appreciable effect on the resistance of the bacilli. There was no apparent relation between the ability of a medium to support the growth of small numbers of tubercle bacilli and thermal lability or resistance to acetic acid. The use of varying types of media probably accounts for the wide variations in thermal resistance reported in the literature. The thermal survival time of avian tubercle bacilli exceeded that noted for human tubercle bacilli under the same conditions of culture and heat exposure.

THE DETECTION OF ANTIGENIC VARIANTS OF BRUCELLA BY MEANS OF AN OPSONOCYTOPHAGIC TEST. Myrtle Munger and I. Forrest Huddleson. Jour. Bacteriol., xxxv (1938), p. 255.

The most satisfactory method of detecting an antigenic variant culture of *Brucella* is an opsonocytophagic test conducted with citrated normal whole blood from humans or guinea pigs. The bacterial cells of an antigenic variant culture are phagocytized in large numbers by the polymorphonuclear cells in citrated normal whole blood from humans and guinea pigs. Bacterial cells of a normal culture are phagocytized slightly if at all by leukocytes in citrated normal blood of the same species. Antigenic variants of *Brucella* are unsuitable for use in the opsonocytophagic test for detecting specific *Brucella* opsonins in blood of human beings or animals.

THE COURSE OF VIRUS-INDUCED RABBIT PAPILLOMAS AS DETERMINED BY VIRUS, CELLS AND HOST. John G. Kidd. Jour. Exp. Med., lxvii (1938), p. 551.

Factors responsible for the observed differences in the course of virus-induced papillomas of the rabbit have shown that some are referable to the virus, others to the cells and others to host differences. The interplay of these factors sheds much light on the nature of the cell-virus relationship in virus-induced tumors. Retrogression of the rabbit papillomas appears to be consequent on a generalized resistance of host origin elicited by and directed against the proliferating virus-infected cells.

JAPANESE B ENCEPHALITIS VIRUS; ITS DIFFERENTIATION FROM SAINT LOUIS ENCEPHALITIS VIRUS AND RELATIONSHIP TO LOUPING ILL VIRUS. Leslie T. Webster. Jour. Exp. Med., lxvii (1938), p. 609.

Japanese B encephalitis virus obtained from Japanese investigators proved virulent for mice and monkeys. It was also found virulent for monkeys when instilled intranasally and for sheep when introduced intracerebrally or intranasally. Japanese B encephalitis virus was differentiated from Saint Louis virus and found similar to louping ill virus according to its reactions in animal species. Serologically it is distinct. Japanese B encephalitis and its related groups of primary virus encephalitides of man are discussed with regard to their differentiation and mode of spread.

A STUDY OF THE MACROPHAGE REACTION IN THE PULMONARY LESIONS OF DOGS WITH EXPERIMENTAL PNEUMOCOCCUS LOBAR PNEUMONIA. O. H. Robertson and Clayton G. Loosli. Jour. Exp. Med., lxvii (1938), p. 575.

The occurrence of the macrophage reaction in the pulmonary lesions of dogs during the course of experimentally induced pneumococcus lobar pneumonia or dying as a result of the infection was found to be a characteristic transformation accompanying recovery of the fixed tissue cells of the lungs. It was also present in varying degrees in the great majority of fatal instances, provided the animal lived more than 40 hours. The longer the animal survived the more pronounced the macrophage reaction observed in the lung lesion at autopsy. The number of pneumococci in the lesions diminished progressively with the evolution of the cellular change which terminated in resolution of the

pneumonic exudate. Some dogs surviving for four days or more showed practically complete clearing of the pulmonary lesions but succumbed with an overwhelming bacteriemia or empyema, or both. On the other hand, several animals dying with a sterile blood exhibited lesions characterized by little or no macrophage response and the presence of many pneumococci. The recovery from experimental lobar pneumonia in the dogs depends on a dual mechanism consisting of a generalized process which prevents and controls invasion of the blood-stream and a local one by which the lesion is finally freed of the invading micro-organism.

CATTLE AS RESERVOIR HOSTS OF SCHISTOSOMA JAPONICUM IN CHINA. Kuang Wu. Amer. Jour. Hyg., xxvii (1938), p. 290.

Small pieces of liver from cattle were collected at random from several municipal abattoirs in Shanghai between December, 1936, and May, 1937, and examined for the possible presence of *Schistosoma japonicum*. The blood-fluke occurred in 50 of 399 animals; the percentage of infection by months varied from 0.84 to 34.4. Seventy-six of 406 buffaloes harbored the parasite; the percentage of infection varying from 8.3 to 24.2 during monthly periods. Eggs of *S. japonicum* were found in the feces of the infected cattle and adults were found in the mesenteric veins of oxen. *S. japonicum* eggs in semi-solid stools were rendered uninfective after they were kept for six days at about 20° C., which indicates that a large percentage of *S. japonicum* eggs are killed during storage in fertilizer pits.

INFECTION WITH TRICHOMONAS FOETUS IN HEIFERS. Justin Andrews and Fred W. Miller. Amer. Jour. Hyg., xxvii (1938), p. 235.

Forty heifers were studied throughout their first pregnancy cycle with reference to the appearance, persistence and effects of infection with *Trichomonas foetus*. The origin of the trichomonad infections seemed to be most highly associated with the first breeding experience of the heifers. The flagellates were found in the vagina for periods varying from seven to 20 weeks. Most of the infections declined spontaneously in clinical severity and in zoölogical density. Abortion, stillbirth, pyometritis, delayed conception and sterility were definitely associated with venereal trichomoniasis, particularly with the more prolonged infections.

The generative disability observed in the heifers did not appear to be due to impotence or sterility of the bulls employed, to Bang's

or other intercurrent disease, to malnutrition or to mismanagement. Non-fertile heifers served repeatedly by infected bulls did not show evidence of reinfection comparable to their original infection. Bulls serving the heifers studied showed higher breeding efficiency when used on cows in the same herd. The authors conclude that trichomoniasis in heifers is sexually transmitted, is variable in duration, tends to be self-limited, causes serious reproductive disturbances when associated with prolonged infections and that resistance to infection is acquired, resulting in greater breeding efficiency in cows in the same herd.

FURTHER STUDIES ON INTESTINAL EMPHYSEMA OF SWINE. D. F. Eveleth and H. E. Biester. Amer. Jour. Hyg., xxvii (1938), p. 364.

Intestinal emphysema of swine is a gaseous distention of the lymphatics of the intestinal wall and mesentery and can be experimentally produced by dietary deficiency. Whole corn, unpolished rice, skimmilk powder and yeast contain the anti-emphysema factor. Skimmilk powder as 20 per cent of the ration does not protect swine but when increased to 50 per cent protection is furnished. A ration of 5 per cent yeast, 20 per cent skimmilk powder and 75 per cent polished rice protected four of five pigs from emphysema. Rations must contain sufficient vitamin B_1 to support growth of about 0.5 pound per day for the experimental production of intestinal emphysema. Chicks and rats do not develop intestinal emphysema on rations which produce these lesions in swine. The histological study of the parenchymatous organs of swine with experimentally produced emphysema did not show evidence of a toxic substance in polished rice.

A STRAIN OF PLASMODIUM RELICTUM FROM DOVES AND PIGEONS INFECTIVE TO CANARIES AND THE COMMON FOWL. G. Robert Coatney. Amer. Jour. Hyg., xxvii (1938), p. 380.

Plasmodium relictum was isolated from the mourning dove (*Zenaidura macroura carolinensis*) and a common pigeon (*Columba livia*). The strains apparently were morphologically the same since each was infective to the other host and the canary. The parasites seemed better adapted to the pigeon than to the dove or the canary. Infections with *Plasmodium relictum* maintained themselves in individual chicks for as long as eleven days, as determined by the smear method, and for as long as 13

days, as determined by subinoculation into the pigeon. Acute infections were maintained in chicks by frequent passage for 23 days.

SULFUR AND RICKETS. C. E. Holmes, H. J. Deobald and C. A. Herrick. Poultry Science, xvii (1938), p. 136.

No evidence of rickets or ill effects from feeding flowers of sulfur was noted if the rations contained as much as 5 per cent flowers of sulfur and in case the chicks had access to direct sunshine. When the ration contained 2, 5 or 10 per cent granular sulfur, "sulfur rickets" resulted when 0.5 or 1.0 per cent cod-liver oil was fed. Two per cent wettable sulfur and 2 to 5 per cent flowers of sulfur fed with 0.5 and 1.0 per cent cod-liver oil also resulted in the development of rickets. When the cod-liver oil was increased to 2 per cent, bone ash increased to normal even though 5 per cent flowers of sulfur or 5 per cent granular sulfur was fed; there was, however, some retardation of growth. The retardation of growth was not so great when the chicks were irradiated 15 minutes daily at 24 inches with a quartz mercury vapor lamp.

ECTODERMAL LESIONS PRODUCED BY THE VIRUS OF ROUS SARCOMA. E. V. Keogh. Brit. Jour. Exp. Path., xix (1938), p. 1.

The virus of Rous sarcoma was propagated for 30 generations on the chorio-allantois of the developing chick. Under the circumstances the virus gives rise to purely ectodermal focal lesions. When inoculated in dilute suspension, mixed mesodermal and ectodermal lesions arise. The enumeration of the discrete lesions may be used as a method of titration of virus. Following inoculation into fowls of emulsions of egg-passage membranes bearing discrete ectodermal lesions, typical Rous sarcomata appear.

A COMPARISON OF THE INTERCHANGE OF THE BODY FLUIDS AFTER INTRAVENOUS INJECTIONS OF CRYSTALLOIDS, GUM ACACIA AND BLOOD-SERUM. Douglas J. Robertson. Brit. Jour. Exp. Path., xix (1938), p. 30.

When hypertonic solutions, such as 33 per cent sodium chloride or 50 per cent glucose, are injected intravenously in cats at the rate of 5 cc per kilo, there is an enormous increase, often reaching a maximum of double the original blood-volume. The increase takes place rapidly and only during the period of injection. The moment the injection was stopped, the blood volume fell rapidly

and in about one-half hour it was back to normal. When 2, 0.9, 0.6, and 0.3 per cent sodium chloride solutions are injected intravenously, fluid leaves the vascular system during the injection so that the blood-volume immediately after the injection is completed is less than the theoretical level expected. The behavior of an intravenous injection of blood-serum is similar to that of either hypotonic, isotonic or 2 per cent solution of sodium chloride. In the mammalian circulation the addition of sodium chloride to gum acacia depresses the power of gum acacia to attract fluid. After a hemorrhage, an injection of blood-serum produces in the blood-volume a similar response to that after an injection of 0.9 per cent sodium chloride and in both the blood-volume rapidly falls after the injection.

LOCAL RECOVERY IN EXPERIMENTAL PNEUMOCOCCUS LOBAR PNEUMONIA IN THE DOG. O. H. Robertson and L. T. Coggeshall. Jour. Exp. Med., lxvii (1938), p. 597.

Six animals showing simultaneous healing and spread of the pathological process in different parts of the lungs were sacrificed during the active stage of the disease for bacteriological and histological study. The resolving lesions were sterile on culture while the young metastatic processes yielded an abundant growth of pneumococci. The resolving areas which represented the older lesions were characterized by the presence of a well developed macrophage reaction, whereas the early lesions consisted of alternating areas of edema and polymorphonuclear infiltration and contained many pneumococci both free and in the cells. The only pneumococci found in the areas of macrophage mobilization were intracellular forms for the most part in the process of digestion. In several instances these striking differences in the cellular picture and the numbers and distribution of pneumococci were observed in different parts of a single lobe lesion. Tests on the blood-serum failed to reveal any evidence of acquired antipneumococcal immunity.

The American Philosophical Society, the oldest scientific organization in the United States, was founded by Benjamin Franklin in 1727.

Diamond Jubilee Convention
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Extensive Publicity Campaign Launched by Associated Serum Producers

A spring advertising campaign to induce more farmers to have their hogs immunized against cholera by their local veterinarians this year, was started last month in fifteen of the nation's largest farm magazines.

The campaign is being sponsored by the Associated Serum Producers, and has been prepared by the American Foundation for Animal Health, the bureau organized by the producers to carry on educational work in the interests of the veterinary profession.

The series of advertisements will run every two weeks through the months of April, May and June, covering the height of the cholera-immunizing season. Each of the advertisements stresses the fact that the only reliable vaccination is that performed by the local veterinarian, and that the farmer who wishes to avoid unnecessary risks should permit no one but his veterinarian to immunize his hogs.

Among the publications carrying the advertising are: *Ohio Farmer, Indiana Farmer's Guide, Weekly Kansas City Star, The Farmer, Prairie Farmer, Nebraska Farmer, Wallaces' Farmer, Chicago Drovers Journal, Omaha Journal-Stockman, Kansas City Drovers-Telegram, Saint Louis Livestock Reporter, Dakota Farmer, Missouri Ruralist, Breeder's Gazette,* and *Successful Farming.*

Backing up their extensive farm paper and newspaper publicity campaign in the interests of the veterinary profession, the Associated Serum Producers this month are releasing an intensive radio campaign over 15 leading stations.

The campaign starts on Indiana and Ohio stations on May 1, and on all other stations on May 16. Primarily, the radio announcements will warn farmers that spring pigs should be immunized against cholera without further delay, and that for safety and protection, the vaccinating should be done only by a veterinarian. At least half the radio campaign will be devoted to

pointing out the importance of the veterinarian's services, and the undue risk involved when the veterinarian is not called in for such professional service.

For those desiring to tune in on the broadcasts in their territories, the stations and broadcasting times are announced as follows:

Station	*City*	*Time*	*Days*
WEW	Saint Louis, Mo.	12:15 p. m.	Mon.-Wed.-Fri.
WROK	Rockford, Ill.	12:50 p. m.	Mon.-Wed.-Fri.
WFBM	Indianapolis, Ind.	12:00 noon	Mon.-Wed.-Fri.
WAAW	Omaha, Neb.	12:13 p. m.	Mon.-Wed.-Fri.
KSOO	Sioux Falls, S. D.	12:30 p. m.	Mon.-Wed.-Fri.
WLS	Chicago, Ill.	7:10 a. m.	Mon.-Wed.-Fri.
WTAX	Springfield, Ill.	12:30 p. m.	Mon.-Wed.-Fri.
KMBC	Kansas City, Mo.	12:05 p. m.	Tues.-Thurs.-Fri.
KFNF	Shenandoah, Iowa	5:59 a. m.	Mon.-Wed.-Fri.
KFAB	Lincoln, Neb.	6:00 a. m.	Mon.-Wed.-Fri.
WBNS	Columbus, Ohio	12:30 p. m.	Mon.-Wed.-Fri.
WHO	Des Moines, Iowa	6:30 a. m.	Mon.-Wed.-Fri.
KSCJ	Sioux City, Iowa	(To be arranged)	
KRNT	Des Moines, Iowa	12:25 p. m.	Mon.-Wed.-Fri.
WMT	Waterloo-Cedar Rapids, Iowa	12:35 p. m.	Mon.-Wed.-Fri.

Kansas State College Student Chapter Holds Dinner-Dance

The annual Dinner-Dance of the Kansas State College Student Chapter of the American Veterinary Medical Association was held in the Wareham Hotel, Manhattan, the evening of April 22. It was one of the brilliant social events of the college year, with an attendance of more than 400 young women and young men college students, as well as visitors and faculty members. The principal address of the evening was made by Dr. D. M. Campbell, of Chicago, editor of *Veterinary Medicine*. His discourse was on "The Value of Veterinary Service to Human Welfare," which he descussed most ably and entertainingly.

The announcement of prize winners in the Division of Veterinary Medicine for the college year 1937-38 was an important feature of the banquet. Each of the following named students was a recipient of a certificate of merit and a cash award:

Physiology

Prizes offered by Dr. N. D. Harwood (K. S. C. '18), of Manhattan, Kan.

*First and Second Prizes..... { Orin E. Ellis, '40 / Roger Guy Spencer, '40

Therapeutics

Prizes offered by Dr. Chas. E. Salsbery (K. C. V. C. '11), of Kansas City, Mo.

First Prize.....Wade Oberlin Brinker, '39

Second Prize.....Guy Arthur Railsback, '39

*Equally divided.

Pathology

Prizes offered by Dr. O. M. Franklin (K. C. V. C. '12), of Amarillo, Texas.
First Prize.....Herald George Wixom, '38
Second Prize.....Jacob Antelyes, '38

Small-Animal Medicine

Prizes offered by Dr. C. W. Bower (K. S. C. '18), of Topeka, Kan.
First Prize.....Charles B. Randall, '38
Second Prize.....Joseph George Feinberg, '38

General Proficiency

Prizes offered by Drs. Benj. F. Pfister (K. S. C. '21) and Earl F. Hoover (K. S. C. '24), both of Kansas City, Mo.
First Prize.....Joseph R. Sterling, '38
Second Prize.....David Jacobson, '38

Phi Zeta at Pennsylvania

The Beta Chapter of Phi Zeta, Honorary Society in Veterinary Medicine, met at the Hotel Sylvania, Philadelphia, April 23, 1938, for the annual initiation of new members from the University of Pennsylvania. The initiates were as follows:

Honorary: Drs. Edward a Rile (U. P. '06) and Gerry B. Schnelle (U. P. '26).
Active: Drs. Samuel F. Scheidy (U. P. '29) and Robert O. Biltz (U. P. '22).
Seniors: Richard G. Buckingham, Edward C. Bulger, Irving G. Cashell, Philip L. Gauntt, Jr., and Armour C. Wood.
Juniors: Howard L. Baker and Samuel H. Moser.

At this meeting, presided over by Dr. William J. Lentz, the following officers were elected: President, Dr. Evan L. Stubbs; vice-president, Dr. Harry C. Campbell; executive officers, Dr. Frank E. Lentz, Dr. A. Henry Craige, Jr., and Howard L. Baker.

Dr. E. L. Stubbs, National President, reported on the recent national meeting held at Omaha, Neb., in August, 1937.

Dr. Clarence J. Marshall delivered an instructive and inspiring address on the subject of prominent alumni of former times. Dr. Marshall stressed in particular the contrasting personalities between Drs. Leonard Pearson and John W. Adams and the manner in which their characters contributed richly in the development of the School of Veterinary Medicine. Having known these men intimately, Dr. Marshall was able to give his address in a delightful personal manner with anecdotes from his own experience.

A. H. C., Jr.

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Regular Army

Captain Robert A. Boyce, Jr., is relieved from further assignment and duty as a student, Army Veterinary School, Army Medical Center, Washington, D. C., effective on or about June 1, 1938, and assigned to Fort Sam Houston, Texas, for duty.

Colonel Robert J. Foster is relieved from further assignment and duty in the office of the Surgeon General, Washington, D. C., effective on May 2, 1938, and is then assigned to duty with the Veterinary Corps, headquarters, Ninth Corps Area, San Francisco, Calif. Colonel Foster will proceed to Brooklyn, N. Y., and report to the commanding general, New York Port of Embarkation, for temporary duty until the sailing of the transport on or about July 16, 1938, for San Francisco, Calif., and will sail on that transport; upon arrival in San Francisco will report to the commanding general Ninth Corps Area, for duty. In addition to this duty, Colonel Foster is assigned to duty as station veterinarian, Presidio of San Francisco, Calif., attending veterinarian at Hamilton Field, Fort McDowell, Letterman General Hospital, and the R. O. T. C. unit of Leland Stanford Jr. University.

First Lieutenant John H. Rust, 3d, is relieved from further assignment and duty at Fort Benning, Ga., effective on or about June 1, 1938, is then assigned to duty at Fort Slocum, N. Y., and will proceed to that station and report to the commanding officer for duty.

Veterinary Reserve Corps

NEW ACCEPTANCES

Rich, Absalom Berry, Jr....1st Lt....Box 855, Austin, Texas.
Rohrer, Raymond Richard...1st Lt....111 Center Sq., Orwigsburg, Pa.
Stiern, Walter Wm.....1st Lt....1729 Owen St., Bakersfield, Calif.
Todd, Lewis Kay.....1st Lt....1121 So. Eye, Tacoma, Wash.

PROMOTIONS

To

Dunlap, Leo Walter.....1st Lt....113 N. 5th St., Zanesville, Ohio.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Holmberg, Gerald W.....1st Lt....Hq. 1st C. A., Boston, Mass.
Dingwall, Stuart M.....1st Lt....Seattle Q. M. Depot, Seattle, Wash.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY WITH CCC

Harden, Chester N.....1st Lt....Woonsocket, R. I.
Higby, Willard C.....1st Lt....Schenectady, N. Y.
Paxton, John D.....Capt.....Sacramento, Calif.
White, Alfred E.....1st Lt....Los Angeles, Calif.

LOUISIANA VETERINARY MEDICAL ASSOCIATION

The seventh annual short course in connection with the annual meeting of the Louisiana Veterinary Medical Association was held at the Louisiana State University, at Baton Rouge, March 2-3, 1938. Dr. W. T. Oglesby, of the University staff, in his capacity as chairman of the Program Committee, had arranged an excellent program for the meeting.

Dr. Frank Collins, president of the Association, called the meeting to order and Dr. James Monroe Smith, president of the University, delivered the address of welcome. Other members of the University faculty who contributed to the program included Drs. C. T. Dowell, director of the Experiment Station, and J. G. Lee, Jr., dean of the College of Agriculture.

Dr. J. K. Northway, of Kingsville, Texas, gave a very interesting talk on "Range Cattle Practice," in which he mentioned a new breed of cattle, the Santa Gertrudes. His talk was illustrated with lantern-slides. At the clinic, Dr. Northway demonstrated his technic of spaying cows and heifers, using two methods. He showed great skill and speed in these operations and it is stated that he can spay as many as 400 a day with proper assistance.

Our Association was honored with a visit by the president of the American Veterinary Medical Association, Dr. O. V. Brumley, who spoke on "Public Health and Veterinary Medicine" and "Recent Knowledge Regarding Rabies." At the banquet, he gave an interesting talk on "Our National Association." All members were glad to hear of the proposed changes in the JOURNAL.

Dr. Chas. W. Bower, a prominent small-animal practitioner, of Topeka, Kan., was on the program for "Small-Animal Therapeutics." He covered this subject very well and he attached much importance to modern methods of keeping records, case reports and so forth. Dr. Bower also conducted a small-animal clinic, which included several operations, diagnoses of skin troubles and reduction of fractures.

The following officers were elected for the ensuing year: President, Dr. C. E. Page; vice-president, Dr. W. A. McDonald, and secretary-treasurer, Dr. C. M. Heflin (reëlected), all of Baton Rouge.

The banquet was held on the roof of the Heidelburg Hotel and was attended by 76 persons, all of whom seemed to enjoy themselves very much.

C. M. HEFLIN, *Secretary-Treasurer.*

ARKANSAS VETERINARY MEDICAL ASSOCIATION

The annual meeting of the Arkansas Veterinary Medical Association was held at the Hotel Eastman, Hot Springs, March 7-8, 1938, with Dr. J. D. Morton, of Pine Bluff, presiding.

Dr. T. M. Dick made his report as secretary and Dr. C. D. Stubbs discussed the possibilities of getting the American Veterinary Medical Association to come to Arkansas for the 1939 meeting. A motion prevailed to invite the A. V. M. A. to meet in Hot Springs next year and another motion was carried instructing Dr. Stubbs to continue his efforts to secure the meeting.

President Morton appointed the following on the Legislative Committee: Drs. Lee T. Railsbach, J. F. Stanford, E. L. Kittrell, Jno. Smith and H. Shull. Dr. C. D. Stubbs was elected as delegate from Arkansas to the A. V. M. A. House of Representatives and Dr. R. W. Williams was selected as alternate.

The address of welcome was delivered by Mr. Kemp, president of the Hot Springs Chamber of Commerce. He strongly offered his support to the Arkansas Association in extending the invitation for the A. V. M. A. to meet in Hot Springs in 1939. He suggested that no other city is so ideally located with hotel and recreational facilities as Hot Springs. He also pointed out the excellent facilities for holding the clinic at Oaklawn Park.

The balance of the morning session was devoted to the literary program. The following papers were presented:

"The 1937 Outbreak of Encephalomyelitis," by Dr. C. C. Hisel, of Oklahoma City, Okla., with discussions by Dr. D. F. Luckey, and Dr. C. L. Campbell, both of Kansas City, Mo., and Dr. H. E. Curry, of Jefferson City, Mo.

"What a Veterinarian Can Do to Help Improve Live Stock in His Community," by Dr. R. W. Williams, of El Dorado.

"The Control of Rabies," by Dr. H. C. Rinehart, of Springfield, Ill.

The meeting adjourned to allow the members to attend the Oaklawn races in the afternoon.

At the Tuesday morning session, the following papers were presented:

"Johne's Disease," by Dr. I. T. Reed, of Dumas.
"The Status of Bang's Disease Eradication in Arkansas," by Dr. A. W. Rice, B. A. I. Inspector-in-Charge, of Little Rock.

Then followed an open discussion on poultry diseases and bot-control work in Arkansas. Dr. C. L. Campbell, of Kansas City, gave a talk on "Newer Conceptions of Virus Diseases." This was illustrated with motion-pictures.

The following officers were elected for the ensuing year: President, Dr. J. D. Morton, of Pine Bluff; first vice-president, Dr. Lee T. Railsbach, of Harrison; second vice-president, Dr. Jno. Smith, of Stuttgart, and secretary-treasurer, Dr. I. T. Reed, of Dumas.

I. T. REED, *Secretary-Treasurer.*

AMERICAN ANIMAL HOSPITAL ASSOCIATION

The American Animal Hospital Association held its fifth annual meeting at the Mayflower Hotel, Washington, D. C., March 24 to 26, 1938. Unusual interest was manifested by those in attendance because of several most interesting features that were presented.

First, a demonstration of the use of a device for sustaining respiration in dogs for thoracic surgery was presented by Dr. Allan C. Secord, of Toronto, Canada. Dr. Secord executed a very fine technic which he has developed for the correction of rupture of the diaphragm in dogs. His operation is done between ribs and it is quite a successful procedure as he performs it. He has found that most diaphragmatic hernias involve the right crus.

Air-conditioning of hospitals for small animals has been made use of considerably during the past year and great interest has been manifested in this subject. Dr. E. Burton Corning, a Washington architect, discussed newer materials employed in the construction of hospitals with special reference to materials that enhance sanitation and increase the comfort of the patients hospitalized. "No element of material, design or structure that has proved its value in human hospitalization is overlooked in planning the modern hospital for animals."

At the evening session on the first day, Dr. Maurice C. Hall, Chief of the Division of Zoölogy, U. S. Public Health Service, Washington, D. C., discussed "Parasites and Parasite Control." Keen interest in Doctor Hall's presentation was manifested by

an appreciative audience when he discussed trichiniasis. He showed why the keeping of records is necessary in any epidemiologic or epizoötological investigation. Incidentally, according to the work of the Division of Zoology, U. S. Public Health Service, approximately 16 or 17 per cent of all Americans are found to harbor trichinae, when they come to the autopsy table.

Studious consideration was given the matter of possible standardization of hospital records by Drs. A. R. Theobald, of Cincinnati, Ohio, and J. A. S. Millar, of Deal, N. J. A well-prepared exhibit of clinical and laboratory records forms contributed by members of the Association covering America served as a basis for this discussion. In addition to the usually ascribed purpose that records serve in hospitals, they are given serious consideration in epizoötological studies as a basis for compiling statistical data on any disease.

Dr. E. B. Dibbell, of Baltimore, Md., presented a report on hospital equipment and its uses. This presentation was illustrated by charts, sketches and specifications. In a few instances working models were presented by many member hospitals. Keen interest was manifested in Doctor Dibbell's presentation of this very practical subject.

Convulsions in dogs of all ages are alarming symptoms that disturb any dog-owner. A paper on "The Significance of Convulsions in Dogs," by Dr. Carl F. Schlotthauer, of the Mayo Foundation, Rochester, Minn., was presented by Dr. Jesse Bollman, of the same institution. In the discussion of causes of convulsions in dogs, Dr. Schlotthauer mentioned causes as being lesions involving the brain, spinal cord or their coverings, injuries of various kinds, infectious diseases and their complications and intestinal parasites.

Dr. M. L. Morris, of New Brunswick, N. J., chairman of the Committee on Foods, reported on the work of the second year, during which its objectives have been carried to a satisfactory point of progress. The completion of tests on some dog foods and the granting of the seal of approval on four foods was announced. At the completion of this report, Dr. H. W. Jakeman, chairman of the Executive Board of the American Veterinary Medical Association, extended greetings from President Brumley and best wishes and congratulations of the A. V. M. A. Dr. Jakeman congratulated the American Animal Hospital Association on its work and assured those in attendance at this meeting that the A. A. H. A. could expect 100 per cent coöperation from the A. V. M. A. in the work of testing and identifying foods.

A prolonged luncheon session for members only was held on the second day, at which time the Secretary and Treasurer gave their reports and officers were elected. Dr. S. W. Haigler, of Saint Louis, Mo., was reëlected president. Dr. L. H. LaFond, of Detroit, Mich., was elected first vice-president. Dr. D. A. Eastman, of Moline, Ill. was reëlected secretary and Dr. A. R. Theobald, of Cincinnati, Ohio, was reëlected treasurer. Dr. C. P. Zepp, of New York City, was elected a member of the Executive Board and Dr. J. V. Lacroix, of Evanston, Ill., was made Director of Public Relations and chairman of the Committee on Publicity.

The afternoon session of the second day was given to the subject of intestinal surgery. Dr. Victor Carraba, of the staff of the New York University Hospital, ably presented the subject, featuring those operations which were found practical and necessary in canine surgery, after which he demonstrated the principles that had been discussed.

The evening session consisted of a dinner and entertainment at the Mayflower Hotel.

"Kidney Dysfunction in the Dog and Its Relations to Sulfanilamide Therapy" was presented by Dr. James B. Allison, of the Department of Physiology and Biochemistry, Rutgers University. This paper was the result of the work of Dr. David F. Green, of the same department, and Dr. Mark L. Morris, of Stelton, N. J. The report reflected a cautious exploration of the application of sulfanilamide in the treatment of certain diseases of dogs. It was discussed by Dr. Bollman.

A paper on "Infectious Gastritis of Canines" was presented by Dr. H. J. Shore, of Fort Dodge, Iowa. In this paper the author presented evidence of the existence of an enteritis in dogs that could be reproduced by inoculations of cultural material or by feeding cultures to dogs. The clinical symptoms of this type of gastro-enteritis simulate those that signalize Stuttgart disease and Weil's disease in several respects.

Dr. Roger S. Amadon of the University of Pennsylvania, presented a very complete discussion on "Some Phases of the Pathological Physiology of the Blood-Vascular System of the Dog."

DISTRICT OF COLUMBIA VETERINARY MEDICAL ASSOCIATION

The second quarterly meeting of the District of Columbia Veterinary Medical Association was held in the Mezzanine Room of the Mayflower Hotel, Washington, on April 12, 1938. The

meeting was well attended and veterinarians from the adjoining states and Pennsylvania were guests.

At this meeting President Weadon presented Lt. Col. R. A. Kelser, a native of Washington, who had just returned to the States from the Panama Canal Zone to become Director of the Veterinary Corps of the United States Army. Lt. Col. Kelser gave a very instructive discussion on the subject, "Host and Tissue Adaptations to Filtrable Viruses." Col. Kelser's remarks dealt principally with the latest findings of research workers concerning the character and behavior of viruses and their adaptability to certain specific tissues.

At a meeting held previous to the one above mentioned, the following officers were reëlected for the ensuing year: President, Dr. Mason Weadon; first vice-president, Dr. H. W. Schoening; second vice-president, Lt. Col. G. L. Caldwell, and secretary-treasurer, Dr. E. Barnwell Smith.

W. M. MOHLER,
A. V. M. A. Res. Sec. for the District of Columbia

Thirteenth International Veterinary Congress

The following is the first installment of the list of veterinarians in the United States who have filed their applications for membership in the Thirteenth International Veterinary Congress to be held at Zurich-Interlaken, Switzerland, August 20-27, 1938. Additional installments of the list will be published during the coming months.

Brumley, Oscar V., Columbus, Ohio.
Campbell, D. M., Chicago, Ill.
Christy, C. M., Brookville, Pa.
Crossman, E. A., Needham, Mass.
Darby, Howard L., Fort Worth, Texas.
Dennis, William A., Jamestown, N. Y.
Dick, G. A., Philadelphia, Pa.
Edelin, A. A., Baltimore, Md.
Ferguson, Thomas H., Lake Geneva, Wis.
Haring, C. M., Berkeley, Calif.
Hart, George H., Davis, Calif.
Hoskins, H. Preston, Chicago, Ill.
Huebner, Richard A., Philadelphia, Pa.
Joss, Edward C., Washington, D. C.
Jungherr, Erwin L., Storrs, Conn.
Kinsley, A. T., Kansas City, Mo.
Lacroix, J. V., Evanston, Ill.
MacKellar, W. M., Washington, D. C.
Marshall, C. J., Philadelphia, Pa.
Millar, Joseph A. S., Deal, N. J.
Miller, A. W., Washington, D. C.
Miller, F. W., Silver Spring, Md.
Pace, John C., Calexico, Calif.
Schlingman, A. S., Detroit, Mich.
Strange, C. Roy, Madison, Wis.
Wegner, E. E., Pullman, Wash.
Wight, A. E., Washington, D. C.

NECROLOGY

CHRISTIAN ROTHAUG

Dr. Christian Rothaug, of Sea Cliff, Long Island, N. Y., died November 3, 1937. Heart trouble with other complications was the cause of death.

Born in Brooklyn, N. Y., July 31, 1873, Dr. Rothaug was graduated from the American Veterinary College, class of 1896. He served on the staff of the Brooklyn Board of Health for a number of years, but was engaged in private practice at the time of his death.

Dr. Rothaug joined the A. V. M. A. in 1918. He had been a member of the Veterinary Medical Association of New York City at one time.

CARL J. WALLEN

Dr. Carl J. Wallen died at the Veterans' Hospital, Sawtelle, Calif., on December 19, 1937. Death apparently was due to peritonitis caused by an abscess that had formed around a bone graft on the spine that had been performed in 1917. The abscess had ruptured into the peritoneal cavity.

Born in China, March 1, 1894, Dr. Wallen received the equivalent of a high school education and enlisted in the United States Navy in the early part of 1917. Shortly thereafter, he was in an explosion on one of the U. S. battleships and was severely injured, necessitating the amputation of one leg in addition to the bone graft already mentioned. He was discharged from the Navy in November, 1917, with complete disability.

Later, Dr. Wallen entered Ohio State University for the study of veterinary medicine. Following his graduation in 1923, he accepted a position with the California State Department of Agriculture. Later, he returned to his Alma Mater for graduate work and received a Master's degree. He returned to California and resumed work with the State Department of Agriculture. In 1934, he accepted a position as veterinarian to the Puritan

Poultry Corporation at Atascadero. He held this position until the latter part of 1936, when he opened an office in Glendale to engage in poultry practice.

Dr. Wallen joined the A. V. M. A. in 1924. He is survived by his widow, one son, one daughter, one brother and two sisters.

W. L. C.

WILLIAM L. BLEECKER

Dr. William L. Bleecker, of Waterloo, Wis., died suddenly at his home, February 28, 1938, following a heart atttack.

Born at Hubbleton, Wis., April 9, 1880, he attended the Waterloo High School and was graduated in 1898. In 1900, Dr. Bleecker, with his family, moved to Minnesota. He then entered the School of Agriculture of the University of Minnesota. Following his graduation he continued to live in Minneapolis. In 1908 he went to Mississippi, where he was engaged in dairy extension work for four years. He then decided to study veterinary medicine and entered the Ohio State University. Following his graduation with the class of 1917, he engaged in general practice at Sidney, Mont., for one year and then accepted a position in the Veterinary Division at the University of Arkansas, Fayetteville. He remained there for 15 years, until poor health forced him to resign. He then returned to Waterloo and continued to live there until his death.

Dr. Bleecker joined the A. V. M. A. in 1921. He was a member of the Society of American Bacteriologists and was a Past Master of Washington Lodge, F. and A. M., of Fayetteville. He is survived by his widow (née Margaret Legge), two brothers, one of whom is Dr. D. E. Bleecker (Chi. '09), of Waterloo, and two sisters.

FRANK S. BEATTIE

Dr. Frank S. Beattie, of Iola, Kan., died March 17, 1938, at the age of 74 years.

Born at Seaforth, Ontario, Canada, Dr. Beattie was graduated from the Ontario Veterinary College, class of 1889. Before moving to Iola, where he had been a resident for 35 years, as well as a civic leader, he had lived in Detroit, Mich., where he was connected with the old Home Telephone Company.

Dr. Beattie joined the A. V. M. A. in 1918. He is survived by three sisters and one brother.

WILLIAM M. KINNEY

Dr. William M. Kinney, of Wooster, Ohio, died at his home, March 12, 1938, following an illness of several months. He was 71 years of age and was reported to have been Wooster's oldest veterinary surgeon. He was a graduate of the Ontario Veterinary College, class of 1892, and had been in active practice in his community for 45 years. He is survived by his wife, four sisters and two brothers.

BERNARD FRANCIS REINHARD

Dr. Bernard F. Reinhard, of Saint Cloud, Minn., died at his home on March 25, 1938. Parenchymatous nephritis was given as the cause of death.

Born in Saint Cloud, Minn., April 16, 1882, Dr. Reinhard was graduated from the School of Pharmacy, University of Minnesota, in 1905, and from the Chicago Veterinary College, class of 1909. Following his graduation, he engaged in the practice of veterinary medicine at Saint Cloud, and for the past six years held the position of City Dairy Inspector.

Dr. Reinhard was a member of the Minnesota State Veterinary Medical Society. He is survived by two sisters.

J. P. F.

MILES LEROY DAVENPORT

Dr. Miles L. Davenport, of Saint Paul, Minn., died of a heart ailment on April 10, 1938.

Born in Nanticoke, Pa., July 26, 1869, Dr. Davenport was graduated from Alfred University in 1898, with the degree of B. S. He then entered Cornell University for the study of veterinary medicine and was graduated in 1903. Following his graduation, he practiced veterinary medicine in New York. Later, he entered the service of the U. S. Bureau of Animal Industry and was stationed at Milwaukee, Wis., and Chicago, Ill. In 1910, he became inspector-in-charge at Fergus Falls, Minn., which position he held until his retirement in 1932. He then moved to Saint Paul where he remained until his death.

Dr. Davenport joined the A. V. M. A. in 1910. He was a member of the Minnesota State Veterinary Medical Society. He was a Knight Templar and a member of the Royal Arch Masons, Chapter 21. He is survived by his widow (née Cora LeJeune), one daughter, four sons, one sister and one brother.

J. P. F.

LEVI UPTON SHIPLEY

Dr. L. U. Shipley, of Sheldon, Iowa, died on April 16, 1938. He had been in poor health for several months, but his death came suddenly, following a few days of confinement in bed.

Born August 24, 1863, at Magnolia, Ill., Dr. Shipley received his veterinary education at the Chicago Veterinary College. Following his graduation in 1892, he located at Sheldon, Iowa, for general practice. He soon established a reputation as one of the best surgeons in northwest Iowa. He could recall many interesting trips in consultation with other practitioners, and many veterinarians of the younger generation can trace their inspiration and qualifications to their associations with him. In 1917, Dr. Shipley became associated with Dr. W. H. Hurst (Iowa '02) and the partnership of Drs. Shipley and Hurst endured to the end.

Deeply interested in public service, Dr. Shipley had served his city, in one capacity or another, for a period of ten years. He was a member of the city council from 1922 to 1924. Following this, he was mayor for four years. He retired from politics for a few years, but returned in 1934 and served two terms as councilman-at-large. He had declined to be a candidate for reëlection within a month before his death.

Dr. Shipley joined the A. V. M. A. in 1905 and at the time of his death was among the oldest members on the roll. He was a member of the Iowa Veterinary Medical Association, was a past president and, at some time during his career, had served on practically every committee of the organization. Neither erratic nor sensational in his conduct, his counsel and advice were always sound. He was the physical embodiment of the old axiom that "still runs the water where the brook is deep."

Dr. Shipley was a member of Mistleto Lodge, F. and A. M., and a member and Past Worthy Patron of the Order of Eastern Star, of Sheldon. He is survived by one daughter, Lora, who had been his constant companion since the death of his wife in 1922. A son died at an early age. Between 25 and 30 veterinarians attended the funeral service at the Methodist Episcopal Church, in Sheldon, on April 19.

J. P.

Our sympathy goes out to Dr. J. T. Molison, of Mount Gilead, Ohio, in the death of his wife, Mrs. Emma F. Molison, on February 20, 1938, after an illness of two days; and to Dr. W. D.

Staples, of Anniston, Ala., in the death of his wife, Mrs. Luelle Allen Staples, on April 22, 1938, after an illness of about six weeks.

Virginia Governor Vetoes Undesirable Legislation

At the 1938 session of the Virginia State Legislature, Senate Bill 103 was introduced, designed to amend and reënact Section 1273 of the Code of Virginia, relating to the Veterinary Practice Act. The proposed amendment would have given power to county agents, their assistants and game wardens, to use vaccines on animals in counties where there is no registered veterinarian. The amendment was passed by both bodies of the General Assembly by a very small margin. However, through the energetic efforts of the Legislative Committee of the Virginia State Veterinary Medical Association, the governor was prevailed upon to veto the bill. Through the very commendable action of the governor, this undesirable piece of legislation was not placed upon the statute books of the Old Dominion.

PERSONALS

MARRIAGE

Dr. Benjamin Rosenfeld (Iowa '37), of Osage, Iowa, to Miss Averill Wright, of Ottumwa, Iowa, April 8, 1938.

BIRTHS

To Dr. and Mrs. Henry F. Winiecki, of Sioux City, Iowa, a daughter, Kathryn Elaine, April 12, 1938.

To Dr. and Mrs. Horace D. Pritchett, of Philadelphia, Pa., a daughter, Carolyn Scott, April 19, 1938.

PERSONALS

Dr. R. W. Jackson (St. Jos. '20) has removed from Frenchtown to Clinton, N. J.

Dr. G. F. Motteler (Wash. '36), formerly located in Lewiston, Idaho, is now in Colville, Wash.

Dr. A. J. Sirilo (O. S. U. '34), formerly of El Cerrito, Calif., reports a new address: Windsor, Ohio.

Dr. L. A. Dibert (Chi. '11), of Cullom, Ill., has resumed general practice, after a long siege of illness.

Dr. William J. Seagers (Corn. '35) changed his address, March 1, from Binghamton, N. Y., to Knoxville, Pa.

Dr. Harvey L. Fell (U. P. '25) has opened a hospital for small animals at 1101 North Clayton St., Wilmington, Del.

DR. L. A. MOSHER (Mich. '15), of Atlanta, Ga., spent part of the winter in Key West, Fla., in the interests of his health.

DR. THOMAS E. RANDALL (Cin. '17), formerly on the staff of the State Veterinarian, has located at Albany, Ky., for general practice.

DR. JAMES DEUBLER (A. P. I. '35) is now associated with Dr. T. W. Brown (A. P. I. '35), in general practice at Greeneville, Tenn.

DR. EDWARD D. MARTIN (O. S. U. '11) is now located at the State Racoon Farm, at Milan, Ohio, with the Division of Conservation.

DR. G. W. ROBSON (O. S. U. '30), of Mount Victory, Ohio, has purchased the practice of Dr. Bernard H. Gibson (O. S. U. '35), at Newark, Ohio.

DR. W. M. RUSH (S. W. '13) has accepted a position as veterinarian to the Texas State Prison System and is now located at Huntsville, Texas.

DR. J. H. BINNIG (O. S. U. '34) has resigned his position with the U. S. Bureau of Animal Industry and has located at Minerva, Ohio, for general practice.

DR. ROY L. SCOTT (Ind. '15), of Greenwood, Ind., is mentioned in local papers as a candidate on the Republican ticket for the office of trustee of Pleasant Township.

DR. C. C. HARROLD (Ind. '12), of Frankton, Ind., announced his candidacy for the nomination of County Clerk on the Republican ticket, the early part of April.

DR. JOSEPH W. STULTS (McK. '11) was retired from active service in the U. S. Bureau of Animal Industry, on January 31, 1938. He was stationed in Oklahoma City.

DR. F. E. BROAD (McK. '17) has returned to his practice in Plymouth, Ind., after more than three years of active service in the Veterinary Reserve Corps of the Army.

DR. J. R. STIFLER (U. S. C. V. S. '11), of Lebanon, Ky., who was injured in an automobile accident several months ago, has recovered sufficiently to resume practice.

DR. ANDREW KOOSTRA (Iowa '38) has located at Bowling Green, Ky., for general practice. He formerly was supervisor of the Blue Grass Dairy Herd Improvement Association.

DR. CARL R. BENTON (Mich. '36), who has been with Dr. A. H. Russell (Corn. '20), of Concord, Mass., for over a year, has entered practice on his own account at Salem, Mass.

DR. A. E. ERICKSON (Gr. Rap. '18), of Charlotte, Mich., addressed the local chapter of the Future Farmers of America on April 5. His subject was "The Prevention of Animal Diseases."

DR. MANLEY MATHERS (Chi. '15), of Mason City, Ill., addressed the students of the Illinois State Normal University, at Bloomington, on April 8. His subject was "The Care of the Horse."

DR. STANLEY M. KRUKOWSKI (Corn. '34) has terminated his active service with the Veterinary Reserve Corps and has accepted a position with the Borden's Farm Products Co., at Whitehall, N. Y.

DR. M. J. HUGGINS (Chi. '13), of Edwardsville, Ill., addressed the Highland (Ill.) Rotary Club on April 13. His topic, "The Forgotten Man," was an interesting presentation of the rôle of the modern veterinarian.

DR. ALANSON L. ALBRIGHT (McK. '03), of Cayuga, Ind., has announced his candidacy for the nomination for Representative in Congress, from the Sixth District, on the Democratic ticket. He was elected to the State Senate in 1932.